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RECENT ADVANCES IN PULMONARY TUBERCULOSIS

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PREFACE

TUBERCULOSIS has been one of the great problems of medicine since the earliest days of history. It is only natural in such a disease that many remedies should have been recommended, but their vogue has usually been of very short duration, and it has been wisely said that when a new " cure " for consumption appears those who want to use it should do so at once before it goes out of fashion and ceases to operate.

In writing this book, the author has tried to describe the recent increase in our knowledge of tuberculosis, and to separate the real advances in treatment from those methods which have no scientific basis and enjoy but brief, though occasionally recurrent, phases of popularity.

Most of the book deals with treatment, and no attempt has been made to describe physical signs or morbid anatomy, because the work is intended for practitioners and senior students, and not as an examination text-book. Chapters on diagnosis and prognosis are included, and an introductory chapter describes certain animal experiments, with modern views as to immunity and spread of disease, for treatment is largely based on these views.

The author wishes to thank Dr. Davies, of Cefn Mably Sanatorium, for permission to reproduce the skiagram of silicosis, and Dr. Stanley Melville for all the other skiagrams in the book.

L. S. T. BURRELL

AUG 13 1937

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RECENT ADVANCES IN PULMONARY TUBERCULOSIS

CHAPTER I

INTRODUCTION

Although tuberculosis has been one of the most important medical problems since the earliest ages, fifty years have not yet passed since the discovery of the tubercle bacilli by Koch. Not only did this discovery give great impetus to research, which led to a rapid development of our knowledge of tuberculosis, but our views on many of the problems connected with the subject have undergone a complete revolution since that time.

For example, we now know that the enormous majority of individuals in a civilised community are, or have been, infected by tubercle bacilli; we also know that this infection gives a certain degree of protection against the spread of the disease. If we take a positive tuberculin test as indicating the presence of a tuberculous lesion in the body, are we to regard this as favourable because the individual has that protection which is lacking in one who shows a negative result to the tuberculin test? But the latter has yet to be infected before he can develop any form of tuberculosis, whereas one who is already infected has in him the seeds which may break out into serious or fatal disease. In other words, can we speak of a "beneficial" and a "harmful" tuberculosis? Again, how great is the protection afforded by previous infection? To what extent is a consumptive a danger to people themselves already infected? In trying

to prevent tuberculosis should our efforts be directed to vaccination or ensuring that every one is mildly infected, or should we try to abolish all possible sources of infection ? It has been suggested that infants obtain protection by drinking tuberculous milk, and some will actually oppose the sterilisation of milk on the grounds that this deprives children of a valuable source of protection.

There are many other problems of vital importance to the treatment and prevention of tuberculosis which have arisen out of the discovery of the tubercle bacillus. It will be well, therefore, to discuss infectivity and immunity before considering modern methods of treatment.

When an animal is inoculated with tubercle bacilli the subsequent course of events depends on whether it is already tuberculous or not. If tubercle bacilli are injected intracutaneously into an animal not previously infected nothing happens for the first few days. Then at the site of the inoculation appears a nodule which gradually increases in size. This nodule is essentially composed of epithelioid cells, the origin of which is uncertain, but which eventually become fibroblasts and lead to a fibrosis around the bacilli. The formation of the tubercle or nodule is not inflammatory, but due to proliferation of the cells, and may be compared to the growth of a neoplasm. Some two or three weeks after inoculation a zone of inflammation appears around the tubercle, which breaks down, forming an ulcer and, at the same time, certain general signs of ill-health develop.

If tubercle bacilli are injected intracutaneously into an animal previously infected the course of events is different. There is an inflammatory exudative reaction within a few hours at the site of the inoculation. There is no latent period as in the case of a first infection. In addition to the local inflammation there may also be focal inflammation around already existing lesions in other parts of the body. If the dose of re-infection is sufficiently large local ulceration may occur, but it tends to heal and any new nodular formation

which occurs appears sooner than in a first infection and tends to subside in the tuberculous animal. Therefore there are two factors in regard to reaction to re-infection.

1. The natural reaction of the non-infected animal. This is proliferative and non-inflammatory.

2. The acquired reaction peculiar to the previously infected animal. This is inflammatory. The tissues of such an animal are hypersensitive to tuberculin or tubercle bacilli, and this condition of hypersensitiveness is spoken of as " allergy." In the case of a first infection no symptoms of ill-health develop until the animal tissues become sensitive to the tuberculous infection, that is, until allergy appears. Immunity is a function of allergy, and it may be said of tuberculosis as has been said of typhoid fever that the beginning of symptoms marks the beginning of the immune period. Large doses of tuberculin which would be fatal to the allergic animal cause no symptom at all in the non-infected one, and the same applies to the injection of tubercle bacilli, except that after a latent period the bacilli will cause a general tuberculous infection. The latent or incubation period may therefore be regarded as the length of time taken by the body to establish immunity.

That immunity is a function of allergy may be shown by experiments. The allergic animal is made acutely ill by the injection of tubercle bacilli, but provided it recovers from the initial reaction it will live much longer than one infected by a similar dose of bacilli for the first time, and in the non-immune or non-infected animal allergy does not exist. Dr. Allen K. Krause (1) describes a series of experiments on the non-immune and the immune guinea-pig designed to work out the paths of transit of the tubercle bacilli. It was found that if the bacilli are introduced by inhalation the first anatomical signs of infection appear in the tracheo-bronchial glands. If inoculated subcutaneously the bacilli are found earlier and in larger numbers in the tracheo-bronchial glands than in the lungs. Indeed, if they are of low virulence they

may not produce any anatomical lesion in the lungs at all. It was also found that the transit of the bacilli was very rapid in the non-immune but delayed in the immune animals. The experiments were very carefully carried out, the following procedure being adopted. A series of guinea-pigs was immunised by infection with a weak strain of tubercle bacilli, and subsequently re-infected with a virulent strain ; at the same time a similar dose of the virulent strain was inoculated into the same number of non-immune guinea-pigs for the purpose of control. The guinea-pigs were then killed at varying intervals after infection and certain organs removed with the greatest precautions against sepsis or cross infection. Each organ thus removed was macerated in a sterile mortar with a little saline solution and injected into the groin of a normal guinea-pig. The organs removed were the iliac lymph node, the spleen, the tracheo-bronchial lymph nodes and the lungs. As a result of these experiments it was shown that in the non-immune animals the tubercle bacilli were disseminated throughout the body in seven days or less. Usually in from three to five days. The local iliac nodes were always infected within three days.

In the immune animals the iliac lymph nodes may contain a few bacilli after three days but not until ten days are they regularly infected, and it may be as much as thirty days before the bacilli are disseminated throughout the body. Two immune and two non-immune animals were killed at intervals varying from three to forty days after infection, and the table on p. 5 shows the times at which the different organs caused tuberculosis when inoculated into a normal guinea-pig.

Not only was it found that there is a delay in the transit of the bacilli in the immune animals, but it was also discovered that when the viscera are invaded the number of bacilli are few for a considerable period in the immunes as compared with the non-immunes.

It is known that the rate of spread of tubercle bacilli from

Days.	I.L.N.		Sp.		L.		T.B.G.	
	I.	N.I.	I.	N.I.	I.	N.I.	I.	N.I.
3	— +	+ +	— —	— +	— —	— —	— —	— —
5	— +	+ +	— —	— +	— —	— +	— —	— —
7	— +	+ +	— —	— +	— —	— +	— —	— +
10	+ +	+ +	— +	+ +	— —	+ +	— —	+ +
13	+ +	+ +	— +	+ +	— +	+ +	— —	+ +
17	+ +	+ +	— +	+ +	— +	+ +	— +	+ +
21	+ +	+ +	+ +	+ +	— +	+ +	— +	+ +
31	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +
40	+ +	+ +	+ +	+ +	+ +	+ +	+ +	+ +

I.L.N.—Ileo-lymph nodes. Sp.—Spleen. L.—Lungs.
T.B.G.—Tracheo-bronchial glands.
I.—Immune. N.I.—Non-immune. +.—Infected.
—.—Non-infected.

the site of infection to internal organs depends on the quantity or dose of infection both in immunes and non-immunes. It is not unreasonable, therefore, to suggest that some immuno-lysin or substances form in the body of the immune animal and by killing a number of the bacilli not only reduce their number but retard their rate of transit. There is some evidence to support this view, but there are also many objections to it. There is no bacteriolytic action on tubercle bacilli treated with serum of normal or immune animals, nor is there any definite experimental evidence of the existence of immuno-lysins. No success has followed the numerous attempts that have been made to treat tuberculous patients with immune serum. There are probably substances in the body tissues which are antagonistic to the growth of tubercle bacilli, but there is no satisfactory evidence of the presence of any such substance peculiar to the immune state. It is well known that cultures of the bacilli if kept in one tube for more than a few months cease to

grow and cannot be subcultured, and it is quite possible that bacilli which have been fixed in one part of the body for a considerable time lose their power of development. Living tubercle bacilli may be found in an old lesion which has ceased to spread and has apparently been healed for many years. Yet in order to live they must have oxygen and nourishment from the body, and therefore be open to action by any immuno-lysin should it exist.

It would seem that some other factor is concerned in fixing the bacilli and retarding their rate of transit through the body, and the zone of inflammation at the site of re-infection in an allergic animal is probably of importance in this respect.

Eventually immune animals which have been re-infected die of generalised tuberculosis but they live much longer. In Dr. Krause's experiments one lived for ten and another for eleven months. The important fact is that re-infection can take place and lead to a fatal result. In the immune a larger dose will be required to cause disease than in the non-immune, but given a sufficient dose the barriers of defence will be broken down.

In the human subject, if the tuberculous lung is collapsed by artificial pneumothorax, the spread of the disease is stopped or, at any rate, retarded. This often gives time for satisfactory barriers of defence to form and leads to permanent arrest of the disease ; on the other hand it may be that the disease will continue to spread, although much more slowly. It is now generally admitted by those with experience of the subject that artificial pneumothorax does prolong life, but one great criticism of the treatment is that the late results are disappointing compared with the initial ones, for it so frequently happens that the disease continues to spread and does in the end prove fatal, and that, therefore, the treatment cannot be considered a cure. Of course it is not a cure any more than is immunity resulting from a previous infection. In Dr. Krause's experiments, to which I have already

referred, all the non-immune guinea-pigs had extensive generalised tuberculosis at the sixty-ninth day, but in an immune one killed at the same time no visible visceral tuberculosis was found, although the spleen gave a positive result on inoculation. It would appear therefore that, in the human subject as in the experimental animal, if the rate of spread of disease is reduced the subject can form barriers of defence which may be sufficient to control the disease altogether, and in any case will delay its progress. The efficiency of the defence must depend on the degree of resistance offered to the spread of the bacilli and also to the quantity or dosage of the infection.

It has been noticed that in the human subject there are two main types of disease. One an acute generalised tuberculosis in which the lungs are affected only as part of a general infection, and this type is seen characteristically in the infant ; and another type which is characteristic of the adult and consists of chronic pulmonary lesions often without any tuberculosis in other parts of the body. In childhood, or the period between infancy and puberty, the human subject is particularly free from all forms of tuberculosis. In order to explain these facts it has been suggested that the acute disease of infancy is due to first infection and may be compared to the results of inoculation in a normal or non-immune guinea-pig, and that the surviving infants have great immunity which renders them free from disease during childhood but gradually wanes and leaves them susceptible to disease modified by previous infection. One would expect immunity to become less and less with time, and tuberculin tests confirm this view. A child with tuberculous glands or joints may give a strongly positive tuberculin reaction, but if the lesions heal it will be found that the reaction to tuberculin becomes weaker as time goes on and may eventually disappear. Again, it is pointed out that if a native of some country or tribe where tuberculosis is unknown is brought into a modern civilised community he is liable to contract

tuberculosis, which tends to run the acute course characteristic of the infant or the guinea-pig, and is probably due to lack of acquired immunity. There is no inherent lack of resistance in the infant, for the enormous majority of them recover without any obvious illness at all although tuberculin tests show that infection has taken place. When there is obvious disease, as indicated by glandular or bone tuberculosis, the child shows a considerable degree of resistance and recovery is the rule.

Animal experiments fail to show that the young of any species is less resistant than the adult. To suggest that a child is liable to " catch " tuberculosis, but that an adult is not so liable, is misleading, although it is true that a larger proportion of adults than children have acquired partial immunity, and, secondly, that a dose of infection which will prove fatal to a child of a few pounds' weight may well be resisted in the case of an adult. It is certainly true that chronic fibroid tuberculosis of the lung is very rare in childhood, but chronic bone or glandular lesions are common, and it is difficult to explain this on the assumption of lack of immunity in the child. Dr. Krause (2) was struck by the fact that in the case of the rabbit infected by tubercle bacilli the early anatomical changes were not the same as in the guinea-pig. In the rabbit the lungs are chiefly affected, whereas in the guinea-pig the lungs frequently showed no obvious disease although the tracheo-bronchial glands were seriously affected. Working on this subject he found that in the guinea-pig there is very little lymphatic tissue in the lung, and that what there is is supplied by the bronchial arteries, whereas in the rabbit's lung there is considerable lymphatic tissue supplied by the pulmonary arteries. Now in the infant there is very little lymphatic tissue but large lymphatic channels in the lungs, whereas in the adult lymphatic tissue has formed and there is not nearly such free lymphatic drainage. May not these anatomical differences account in some degree for the different types of chronic

tuberculosis characteristic of adult and child life ? After all the majority of children do " fix " the invading bacilli, not in the lungs it is true, but in glands, bone, etc., and this can hardly be explained by assuming lack of immunity. In childhood milk is the chief diet, and bovine infection is less uncommon than in adults, but even in infancy, except when the cervical or abdominal glands are involved, most of the cases are due to the human tubercle bacillus. Moreover, even if infection does occur by feeding, the bacilli must leave the intestinal tract by the lymphatic or blood-stream and find their way into the right side of the heart and pulmonary system in order to get into the tracheo-bronchial glands. In other words, they must have passed through the lungs in order to reach the glands where they become fixed.

The site of the lesion is a matter of great importance, tuberculous meningitis is fatal because of its localisation. Tuberculous pleural effusion is followed by pulmonary tuberculosis in a considerable proportion of cases, although several years may elapse before signs of disease appear in the lungs. So-called surgical tuberculosis very rarely spreads to the lungs, indeed some think that an old lesion, for example, in the glands, is a protection against the subsequent development of tuberculosis. Tuberculous glands of the neck may rupture externally and cause no impairment of the general health, but should glands happen to rupture into certain parts of the body, for example, mediastinal or retroperitoneal glands, a rapid and fatal spread of the disease may follow. This does not indicate that the patient who dies of tuberculous meningitis is less resistant than the one who recovers from tuberculous peritonitis, or that one with tuberculous adenitis lacks resistance because the glands happen to rupture into an unfavourable position.

The following table shows how very striking is the difference between tuberculosis in adult life and in infancy.

TABLE 1.—*Mortality Table for England and Wales, 1927.*

Ages.	All Forms of Tuberculosis.	Tuberculosis of Respiratory System.
0—1	718	91
1—5	2,284	378
30—35	3,485	3,208

Yet it will be noted that in infancy the mortality in both cases is less than in adult life in spite of immunity. Mortality tables show that after the fifth year of life there is a great drop in the death rate from tuberculosis, but there are two far more remarkable facts—one the sudden increase in mortality which takes place about the fifteenth year of life, and the other that the two sexes are affected very differently. These age and sex variations in the mortality from tuberculosis are common to all countries. In Ireland the amount of tuberculosis and the type differs from that of England and Wales, yet the mortality varies with age and sex in the same way.

TABLE 2 (3).—*Death Rates from Pulmonary Tuberculosis in England and Wales per million living at each age period, Male and Female.*

	AGES.							
	(10–15.)		(15–20.)		(20–25.)		(45–55.)	
	M.	F.	M.	F.	M.	F.	M.	F.
1861–70 .	608	1,050	2,196	3,121	3,894	3,972	3,880	2,865
1871–80 .	483	851	1,685	2,409	3,109	3,154	3,865	2,468
1881–90 .	344	702	1,293	1,809	2,341	2,326	3,505	2,062
1891–1900 .	234	502	995	1,290	1,887	1,591	3,144	1,642
1901–10 .	171	396	756	998	1,521	1,235	2,753	1,310

TABLE 3.—*Mortality under the same Condition for Ireland.*

	Ages. (10–15.)		(15–20.)		(20–25.)		(45–55.)	
	M.	F.	M.	F.	M.	F.	M.	F.
1861–70 .	704	1,109	1,978	2,494	3,311	2,774	3,166	3,144
1871–80 .	632	1,211	2,163	3,102	3,798	3,406	2,304	2,115
1881–90 .	647	1,454	2,287	3,438	4,054	4,103	2,212	2,112
1891–1900 .	567	1,485	2,283	3,410	4,073	3,971	2,299	2,027
1901–10 .	509	1,256	2,004	3,079	3,579	3,571	2,371	1,891

We may take certain facts regarding tuberculosis as established.

1. In the first place the prevailing type of tuberculosis in early child-life is non-pulmonary and frequently acute and general, whereas in adult life it is almost entirely pulmonary, tending to be acute at puberty and early adult life, but more chronic as age advances. These two types closely resemble the types seen experimentally in the non-immune and immune animal. Viewed broadly, one may regard the infant type of disease as the result of an excessive primary infection and the adult type a re-infection or spread of disease, modified by partial immunity resulting from a previous infection which was not sufficient to cause general disease.

2. But the immune child fixes the infection in glands and bones and not in the lungs, although it would appear that the infecting organisms must pass through the lungs. It is difficult to understand how this difference can be due to the acquired immunity. Anatomical differences between the infant and the adult may have some bearing on the problem.

3. Statistics for the last sixty years show that the mortality from tuberculosis has undergone a change, which varies in different countries during different years, but whether the incidence is increasing or decreasing in any particular country there are always two constant factors :—

(1) A considerable increase in mortality as puberty begins.

(2) A drop in mortality as sexual activity declines.

Moreover, this wave of mortality begins earlier and subsides sooner in women than in men.

It would appear, therefore, that during that period of life when sexual functions are active there is something in the body which favours the spread of tuberculosis. It has been suggested that acquired immunity wanes during this period, and this may be the case, but it is not borne out by tuberculin tests, and the type of disease at this period of life is the type seen in the immune animal. In England and Wales in 1927 tuberculosis of all sorts caused :—

2,284	deaths	between	the ages of	1—5.
1,017	,,	,,	,,	5—10.
4,588	,,	,,	,,	20—25.
877	,,	,,	,,	65—70.

Personally, I find it very difficult to accept as an explanation of these facts the suggestion that the initial high mortality in infants is because they are not immune ; that amongst the survivors the mortality becomes halved because they have immunity ; that this immunity wears off so completely, that the mortality becomes more than double that of the maximum in infancy, and then that the immunity again increases until as old age approaches it has reached a high degree of efficiency.

If this is the true explanation the obvious remedy would seem vaccination or some attempt to immunise children as puberty approaches.

4. There are various other factors which influence the incidence of tuberculosis. For example, when a race moves to a foreign country and lives under different conditions, tuberculosis increases. During the Great War the mortality from tuberculosis was doubled and in some countries trebled, but it remained of the adult type seen in the immune animal.

It would seem therefore that immunity remains and that the increased mortality is due to failure of some process or function not of necessity peculiar to the immune animal.

There are, of course, many other factors which influence the type and course of tuberculosis, but I do not propose to discuss them here. I have touched very briefly only on those problems which have a bearing on treatment, and the following conclusions may be drawn from the evidence given in this introduction :—

1. There is a natural reaction to tubercle bacilli and that this leads to the formation of tubercles and, ultimately, fibrosis.

2. That there is also an acquired reaction to tubercle bacilli which is inflammatory and tends to retard the rate of spread of tuberculous infection. This gives time for barriers of defence to arise, and these always delay and may completely arrest the spread of the disease.

3. Re-infection can occur, and adults as well as children can be re-infected.

4. Immunity or lack of immunity does not wholly account for the variations in mortality under different conditions. There are probably some factors in the body which affect the growth or spread of tubercle bacilli and which vary under certain conditions, but which are not necessarily peculiar to the immune state.

5. The site of the infection is of the greatest importance. A degree of tuberculosis which would prove rapidly fatal in one part of the body would produce few, if any, ill effects in another.

REFERENCES.

(1) KRAUSE, ALLEN K. *Am. Rev. Tub.*, 1920. **IV.**, 3; and *ibid.*, 1926, **XVI.**, 3.

(2) KRAUSE, ALLEN K. *Am. Rev. Tub.*, 1926, **XIV.**, 3, 271.

(3) BROWNLEE, JOHN. *Med. Res. Council Special Report*, Series No. 46, p. 76.

CHAPTER II

PREVENTION

THE two main principles of prevention are destruction of infection and increase of resistance in the individual. In countries where there is no tuberculosis the people cannot, of course, become infected, but they fail to get the protection afforded by frequent small infections. Consequently they are extremely vulnerable and liable to contract acute tuberculosis should they move to a district where infection exists. Since tuberculosis abounds in all civilised communities, some will argue that it is wrong to protect children too much from infection, for the tubercle bacilli introduced by milk and other sources go to build up immunity, and so prevent the individuals from developing acute tuberculosis in adult life. Heimbeck (1) found that nurses who have been infected by tuberculosis, as shown by the tuberculin reaction, are less liable to develop clinical tuberculosis than those who have not, or whose infection has so completely disappeared that the tuberculin test is negative. His work suggests that far from a positive tuberculin reaction being a danger signal it is actually a sign of protection, and that a new infection shows its tendency at once to conquer and produce disease or be conquered and establish immunity. If it is conquered, some tubercle bacilli may remain in the tissues and keep alive for years, but they do not tend to become virulent, multiply, and cause disease. Their presence maintains some degree of resistance, and tends to prevent a fresh infection from producing disease. This protection wanes unless it is kept alive by repeated small infections. Heimbeck found that at Oslo 85 per cent. of the children aged nine gave a positive

Von Pirquet reaction, but of 222 military recruits 55 per cent. gave negative results.

Following this school of thought one should advocate increasing the immunity of people by natural and acquired means.

The result desired is that every one should be infected, not sufficiently to develop clinical tuberculosis, but enough to render them immune to the maximum dose of tubercle bacilli they are likely to meet in ordinary life. This, however, is an impossible state to obtain without danger. Even if we admit that a few tubercle bacilli swallowed with milk or inhaled from time to time do protect, we must also admit that an overdose will cause disease. Moreover, a dose that at one time is harmless may prove fatal at another, when the individual's resistance may be lowered by cold, fatigue, or some other condition. It would seem wrong, therefore, deliberately to expose people, and especially children, to infection.

On the other hand, it is pointed out that without infection there can be no disease, and that the chief principle of prevention should be to remove all sources of infection, or at least to dilute the infection as far as possible. Bad ventilation, stagnant air, small rooms, dirt, etc., all tend to concentrate infection, but in a civilised community much can be done to prevent this concentration, and the great drop in the mortality which has followed modern sanitary methods is evidence of their success. The patient with advanced disease who expectorates a large number of virulent bacilli and is too ill to be careful is one of the greatest sources of danger, and such patients should be treated in institutions where proper nursing and precautions against infection are available. If it is impossible to remove the patient the children should be moved from the infected home, and brought up with a family free from tuberculosis. In this country when tuberculosis is diagnosed the case must be notified to the local medical officer of health. In each district is a dispensary with a tuberculosis

officer, who acts as a consultant in doubtful cases and advises as to diagnosis and treatment. The patient is told how to live, or in most cases taught in a sanatorium, so as to minimise the risk of infecting others. The patient's home is visited, and if necessary cleaned and disinfected, the tuberculosis officer examines the members of the family who have come into contact with the patient and keeps them under his observation. Suspicious cases are sent away to convalescent homes in the country, and if the patient, though still infectious, cannot be moved from home the young children are boarded out with some healthy family, so that they do not come in contact with infection.

The Grancher System.—In 1903 Professor Grancher introduced a system in France whereby healthy children were removed from tuberculous infected surroundings. Dr. Armand Delille, of Paris, referred to this system in a paper read at the International Congress on Tuberculosis held in London in July, 1921. He said that the method had met with considerable success, and was not only the most economical, but the most successful method of dealing with the tuberculosis problem. Certainly it would seem more reasonable to prevent children from meeting infection than to inoculate them against all known diseases. The Grancher system is based on the belief that children do not inherit tuberculosis, but acquire it by repeated infections, and it removes the children whilst still healthy from the infected homes and sends them to live with healthy families. This system of boarding out is employed in England and other countries, but greater stress is laid on the delicate children, or those who are said to be mildly affected, the healthy ones generally being left at home. When possible the patient is removed to an institution, thus removing the source of infection and allowing the children to live a normal life at home.

Immunisation.—There are three methods described by Petroff (2).

1. Injecting living virulent tubercle bacilli. He found ten living virulent bacilli could produce tuberculosis in a guinea-pig. The bacilli were counted by Barber's method, and he tried to produce immunity in guinea-pigs (3) by giving gradually increasing numbers of bacilli at weekly intervals, as follows : one, three, five, eight, twelve, and so on. He found that this method gave fairly good immunity, but it was necessary to keep up the injections, and there is a great risk of developing the disease if resistance fails, so that the proceeding was considered too dangerous to employ on human beings.

2. By using avirulent tubercle bacilli. Many attempts have been made to protect by using attenuated bacilli, and the latest preparation is known as B.C.G. This consists of bovine tubercle bacilli, attenuated by 230 passages in thirteen years on potato-glycerin and bile. Petroff's conclusions are that this is a safer method than when virulent bacilli are used, but the end results are not known.

3. By using killed bacilli. He thinks that an increased state of resistance can be produced by injecting dead tubercle bacilli, and, although the immunity is more partial than when living bacilli are used, it is free from danger.

It is generally admitted that the use of virulent tubercle bacilli is too risky, and that tuberculin or dead bacilli when injected do not give an adequate protection. It is, therefore, the attempt to produce immunity by using attenuated bacilli that is receiving most attention at the present moment, and a great deal of work has been done with B.C.G. (Billie-Calmette-Guerin bacillus). Various doubts as to the value and limitations of B.C.G. have been raised, and it has been asked :—

1. Can B.C.G. recover its lost virulence and produce disease ?

If this is possible it is certainly very rare. A very large number of infants have been injected, and, although a few

have become tuberculous, it is quite exceptional. Any lesions which are produced tend to heal.

2. Is it sufficiently virulent to produce any immunity ?

Of course, immunity is always relative, but there is no doubt that a considerable degree of protection is afforded by B.C.G. inoculations. Experiments on calves (4) showed that even a single inoculation of 50 to 100 mg. B.C.G. gave complete protection against 5 mg. virulent bovine tubercle bacilli injected a month later. The experiments on cattle by Lange and Lydtin (5) did not, however, give such favourable results. Calmette observed 982 babies whose inoculation with B.C.G. dated back more than a year and who lived with tuberculous persons, of them only 1 per cent. died of tuberculosis, whereas it was estimated that in Paris 32 per cent. of babies living with tuberculous parents died of tuberculosis. Greenwood (6) criticises these figures and suggests that the mortality from tuberculosis amongst babies living with tuberculous parents is very much less than 30 per cent. Heimbeck (7) states that a considerable proportion of nurses joining the Municipal Hospital at Oslo develop tuberculosis. In 1927 44 nurses giving negative Von Pirquet test, but healthy, and most between the ages of twenty to twenty-five, were inoculated with B.C.G., and a year later not one of them had developed tuberculosis. In 1924, however, 13 out of 51 ; in 1925 17 out of 72, and in 1926 14 out of 62 nurses joining the hospital with negative Von Pirquet reactions developed tuberculosis. Wilbert's (8) experiments on apes went to show that a very satisfactory degree of immunity could be produced by B.C.G., although other workers have failed to obtain such promising results in apes.

When B.C.G. is given by mouth as advocated by some in the case of infants its value is doubtful, and it does not produce a positive Von Pirquet reaction. B.C.G. given subcutaneously, however, does cause the animal or human being to acquire a positive reaction to tuberculin, although the

sensitiveness wears off in many cases, so that in time the subject is again negative to tuberculin.

In spite of varying experimental reports it would seem certain that B.C.G. injected subcutaneously does give relative immunity for a time.

3. For how long does the immunity last ?

This is not known. Fried (9) says that B.C.G. does not cause tuberculosis, but retains its antigenic properties. As long as living bacilli are in the body immunity persists, for there is a struggle between the body and the bacilli. He suggests that immunity in babies lasts about four years.

It has been shown that the most serious age for pulmonary tuberculosis to develop is about puberty. At this time the mortality is higher than at any age including infancy, and since tuberculin tests show that the majority of children at that age have been infected, it would appear that by the time puberty is reached either immunity has waned or some other change in the body has taken place and rendered the immunity insufficient. It would seem that the most important time to increase immunity is just before and during the onset of puberty.

REFERENCES

Prevention.

(1) Heimbeck, J. *Arch. Int. Méd.*, 1928, **XLI.**, 336.
(2) Petroff, S. A. *J.A.M.A.*, 1927, **LXXXIX.**, 285.
(3) Brown, L., Heisse, F. H., and Petroff, S. A. *J.M. Research*, 1914, **XXX.**, 475.
(4) Calmette, A. Guérin C., Weill-Halle, B. *Presse Med.*, 1924, **XXXII.**, 553, and 1925, **XXXIII.**, 825.
(5) Lange, B., Lydtin K. *Zeitsch. f. tuberk.*, 1928, **L.**, 45.
(6) Greenwood, M. *B.M.J.*, 1928, *i.*, 793.
(7) Heimbeck, J. *Bull. Acad. Med.*, 1928, **I.**, 28.
(8) Wilbert, R. *Ann. Instit. Pasteur*, August, 1925, 39, 641.
(9) Fried, B. M. *Boston M. & S. Journ.*, 1927, 197, 488.

CHAPTER III

DIAGNOSIS

The diagnosis of pulmonary tuberculosis is often a matter of great simplicity, and may, indeed, be quite obvious to the non-medical observer. In such cases, however, the patient has usually passed the stage when treatment can be of much value. In the early and doubtful cases the diagnosis is complicated by the fact that many patients who are infected and have evidence (by physical signs, X-ray, tuberculin, or other tests) of old disease are not actually tuberculous in the sense that they have active or spreading disease. Such a patient may be compared to a house in which there has been a fire, but which is no longer on fire. In other words, they do not require treatment for tuberculosis, though they may require treatment for bronchitis, overwork, Graves' disease, or some other condition to which their symptoms are really due. In certain cases a period of rest is required whether or not the symptoms are due to tuberculosis, and there is no better treatment than can be obtained at a sanatorium. Here the rest and routine have a rapidly beneficial effect. In many cases, however, a mistaken diagnosis not only leads to treatment which if not actually harmful is useless, but it fails to give treatment for the actual disability.

It is possible to arrive at an accurate diagnosis if one remembers that no one sign, symptom or laboratory test can be relied on alone, but that the cumulative effect of several signs or symptoms all pointing one way may make a diagnosis certain. For example, loss of weight may be due to diabetes or a variety of conditions, but if other causes are

eliminated and it is associated with changes such as night sweats, cough and evening temperature, it becomes a very valuable piece of evidence in favour of active tuberculosis. The symptoms most often noticed in the early stages are :—

Loss of Energy.—This is very frequently present, but is not often the complaint for which the patient seeks advice, as it is usually attributed to overwork or some other cause, and not taken seriously unless it becomes extreme. The patient feels tired after an ordinary day and is disinclined for any extra activity, such as going out to the theatre ; he is said to be growing lazy or suddenly to have become an old man. When such symptoms are given tuberculosis should be remembered as a possible cause.

Loss of weight is very common in the early stages of active tuberculosis, although some patients get into an advanced stage of the disease without getting thinner. It is especially valuable as a guide to activity, and in chronic cases, when the disease is apparently arrested, a gradual loss of weight is often the first sign of renewed activity.

Cough is almost always present, but it has no special characteristics. As in bronchitis, it is most common on rising in the morning and after meals. It must be remembered that a chronic cough occurs in many other conditions, such as pharyngitis, nasal catarrh or excessive smoking.

Sputum is absent in the early stages, but is rarely absent as the disease progresses. At first it is mucoid, then purulent, and, in the advanced stages, it is mummular.

Tubercle bacilli in the sputum are definite evidence of infection and may be taken as indicating active disease, although there are patients who remain quite well without any evidence of active or spreading disease, but who have tubercle bacilli in the sputum either constantly or periodically. Tubercle bacilli are not often found in the earliest stages of disease, but when the sputum becomes purulent they are almost always present if the disease is due to tuberculosis.

Kingston Fowler (1) reports a series of 188 cases in which tubercle bacilli were found at Midhurst. In these the bacilli were found on the first examination in 167 and on the second in twelve. In one case they were not found until the seventh time. Repeated examinations are necessary, therefore, but at the same time failure to find them after one, or certainly two, examinations is strong evidence against tuberculosis provided the examination is thorough and the sputum is really purulent. If the bacilli are not found a guinea-pig may be inoculated and the injection is best made into the groin. After a week or ten days enlarged inguinal glands will be felt in positive cases. A local swelling appearing after a day or two is due to other organisms and soon subsides, but the subsequent development of an inguinal lump indicates tuberculosis in the great majority of cases. Later, of course, the tuberculosis will spread in the guinea-pig and the diagnosis can be confirmed.

The important point to remember regarding tubercle bacilli is that their absence in definitely purulent sputum, after repeated examination, is very strong evidence indeed against tuberculosis.

Fever.—A certain amount of fever, usually in the evening, is common in cases of active tuberculosis. If the patient is afebrile whilst in bed a rise of temperature may be obtained after he has taken some exercise. It is not sufficient, therefore, to take the temperature only whilst the patient is resting. A definite rise to 99·6° F. or higher after a brisk walk is a suggestive sign.

The range of temperature in pulmonary tuberculosis is an exaggeration of the normal. A rise begins about 2 p.m. and reaches its maximum about 7 p.m. After this it gradually falls until about 5 a.m., when it is subnormal until the afternoon rise. In other words, the characteristic temperature is intermittent, being lowest in the early hours of the morning and highest in the late evening. The greater the activity of the disease the more is the swing exaggerated, and in extreme

cases the temperature is called "hectic," and this usually indicates great activity. A continuous typhoid type of temperature may be found and is of serious import, for it suggests miliary tuberculosis or a general spread of the disease. In some cases the temperature is higher in the morning than at night, this is known as the inverse type. The characteristic type, however, is the important guide to diagnosis in a doubtful case, and if it is not found in the resting patient it may be brought on by exercise.

Night Sweats are present in the early acute stages of pulmonary tuberculosis or during an acute exacerbation in a chronic case. Some patients who sleep with too many bedclothes feel hot during the night and speak of this as having sweats. True night sweats may be very severe, so that the patient has to change his night clothes two or three times in the night. Such cases are very suggestive indeed of acute tuberculosis and most of them have a characteristic smell. These symptoms are rarely present, however, except when the patient has many other signs which leave no doubt as to the true condition, so that they are rarely of great diagnostic value.

Digestive Symptoms.—Loss of appetite and nausea are sometimes, though rarely, the first symptoms noticed. When a patient complains of a gradual impairment of digestive powers the possibility of pulmonary tuberculosis should be borne in mind.

Pleural Effusion.—Unless there is any obvious cause, such as neoplasm, cardiac failure, etc., the development of clear fluid in the pleural cavity may be taken as evidence of tuberculosis. It does not, of course, follow that the lungs are involved, but on the advent of any signs or symptoms suggesting pulmonary tuberculosis a history of pleural effusion strongly supports that diagnosis.

Hæmoptysis.—This may be due to mitral stenosis, bronchiectasis or other causes, but is usually due to tuberculosis and should be assumed to be of this origin unless

there is evidence to the contrary. There may be veins at the back of the throat, but they do not bleed. It may be said that for all practical purposes in cases of hæmoptysis the blood does not come from the back of the throat. Many lives have been lost by the failure to appreciate the enormous importance of hæmoptysis as an early sign in tuberculosis of the lungs.

A **fistula-in-ano** is another condition which should cause serious suspicion.

Physical Examination.—This is very important, but should not by itself determine a diagnosis. In many cases there are signs which are quite consistent with tuberculosis, but to which we must give a different interpretation owing to other facts. For example, apical bronchiectasis, or early carcinoma of lung, may give signs which some would consider pathognomonic of tuberculosis, and the less obvious signs such as altered breath sounds, slight impairment of the percussion note, diminution in Krönig's area of resonance, etc., are present in many conditions. In the survey of school children carried out in Canada (2), 1,392 children were examined physically by X-ray and with tuberculin, and the following conclusions were reached :—

1. Impairment of resonance over any part of the chest may be due to conditions other than tuberculosis.

2. Broncho-vesicular, or still higher pitched breathing, may be present apart from tuberculosis and is normal under conditions of forced breathing.

3. The presence of râles in the chest usually indicates a non-tuberculous infection.

(The authors are referring to school children, and this conclusion must not be taken to apply to the râles heard at the apex of adults. Such râles are usually due to tuberculous disease.)

4. Physical signs (including Eustace Smith and D'Espine signs) pointing to enlargement of the mediastinal glands are present in conditions other than tuberculosis.

I do not want to underrate the value of physical signs, but one frequently meets a patient who has been condemned as tuberculous on the strength of slight impairment of resonance on percussion at one apex, or some such sign, and no other evidence of tuberculosis whatever. There has been a most complete examination of the chest, with full notes of even the minutest detail, yet there are few if any notes on the history, and no examination of sputum, no tuberculin test and no X-ray examination have been made.

Tuberculosis usually affects the upper portions of the lungs, so that disease at or near the apices is probably tuberculous, but a basal lesion is more likely to be non-tuberculous.

Tuberculin Test.—The most satisfactory tuberculin test is the intracutaneous one.

For this purpose four dilutions are prepared. Dilution 1 is made by adding 0·1 c.cm. of Koch's old tuberculin to 0·9 c.cm. of normal saline containing 0·25 per cent. of phenol. Dilution 2 is made by adding 0·5 c.cm. of dilution 1 to 0·5 c.cm. of the diluting fluid. Dilution 3 is made by adding 0·1 c.cm. of dilution 1 to 0·9 c.cm. of the diluting fluid. Dilution 4 is made by adding 0·1 c.cm. of dilution 3 to 0·9 c.cm. of the diluting fluid.

Since 1 c.cm. of O.T. contains 1,000 mg., it follows that :—

0·1 c.cm.	of dilution	1	contains	10 mg. (0·01 c.cm.) O.T.
0·1 c.cm.	,,	2	,,	5 mg.
0·1 c.cm.	,,	3	,,	1 mg.
0·1 c.cm.	,,	4	,,	0·1 mg.

To perform the test the flexor surface of the patient's forearm, just below the elbow, is cleaned with ether and 0·1 c.cm. of dilution 4 is injected intracutaneously with a very fine needle. The injection should not be made subcutaneously, but into the skin, leaving a slightly raised white weal. In the case of children 0·05 c.cm. may be used. If, as often happens, there is no reaction after three days, 0·1 c.cm. of

dilution 3 is injected in the same way, and, failing a reaction, 0·1 c.cm. of dilution 2 is injected three days later. The reaction occurs twenty-four to twenty-eight hours after the injection and consists of a raised area of redness at the site of the inoculation. In very sensitive patients this may go on to vesiculation, or even ulceration, and for this reason it is wise always to begin with the lower dilutions. A positive reaction indicates infection, but not necessarily active disease. It is somewhat curious that this test is employed so little in this country, and there are two chief criticisms of it. First, that since it is positive in cases where the disease is not active it is of little practical use. In other words, a patient with a positive intracutaneous tuberculin test does not of necessity require treatment. This is true, but there is no one symptom, sign or test which does indicate active disease for certain, and a correct diagnosis can be made only by the cumulative weight of evidence. The second objection is that every one except in very early childhood gives a positive reaction. This is a common belief, but is not the case. Out of fifty adult patients sent into my wards at the Brompton Hospital for diagnosis the test was negative in nine, and they were all patients suspected of having tuberculosis. In Dundas (2) (Canada), and the surrounding country, 1,392 children of school age were tested, and of these only 32 per cent. reacted to tuberculin. Dr. Krause (3) writes that in 1927 a skin tuberculin test was made on people in Framingham, and the result was 38 per cent. lower incidence than it was when a similar test was made there in 1917–19, and he points out that this result is consistent with the fact that in New York city the mortality from tuberculosis between 1898 and 1925 declined three times for the general population and six times for infants under two years of age.

A negative tuberculin test is by no means uncommon, and is of the greatest importance in diagnosis. A positive test is of less value in diagnosis as it indicates infection only. Some believe that a condition of activity can be distinguished from

one of quiescence by the severity of the reaction, but it is an undoubted fact that some people give a strongly positive reaction although they have no apparent illness, nor do they subsequently develop any disease, whereas others with definitely active tuberculosis react mildly. At the same time, when a tuberculous lesion heals the sensitiveness to tuberculin does become less as time passes, and a strong reaction does suggest a recent infection or activity. There are several factors, such as intercurrent disease, which blunt a tuberculin reaction, and a course of tuberculin will blunt it or even eliminate it altogether. It would seem unwise, therefore, to set up a fixed standard and say that those who give a reaction of such and such a violence to such and such a dilution have active disease.

Another method of employing the tuberculin test is by the cutaneous method of Von Pirquet. A solution is made consisting of Koch's old tuberculin, 2·5 c.cm., glycerin phenol, 5 per cent., 2·5 c.cm., saline, 5 c.cm.—that is O.T. in a strength of 1 in 4. A drop of this solution is placed on the forearm and a slight scratch made over it. The scratch must be just sufficient to draw blood. If positive an area of redness will appear round the scratch in three days.

Heimbeck (4) applied Von Pirquet's test to girls aged from twenty to twenty-five who were becoming nurses at the Municipal Hospital, Oslo, and in 1924 he found that out of 109 there were 51 negative, 13 of whom subsequently developed clinical tuberculosis, and 58 positive, none of whom developed it. In 1925 he found that out of 114 nurses, 72 were negative, and 17 of these subsequently developed clinical tuberculosis, but only 2 of the 42 positive cases developed it. In 1926, out of 114 cases, 62 were negative, and of these 14 subsequently developed clinical tuberculosis, but only 1 of the 52 positive cases did so.

He also found that after two years' work at the hospital practically all gave a positive result to the Von Pirquet test. Other interesting facts found by Heimbeck are that not only

were about half the nurses negative to the test, but of 79 medical students 47 per cent. and of 222 military recruits 55 per cent. were negative ; but he found 85 per cent. Oslo children at the age of nine positive to the Von Pirquet test.

These findings would suggest the following conclusions :—

1. A positive Von Pirquet test indicates the presence of an infection and probably the presence of actual living bacilli in the body, but it does not indicate that the individual has or will develop clinical tuberculosis ; on the contrary, he is less likely to develop it than a non-reactor.

2. A fresh infection tends quickly to get the upper hand and produce clinical disease, or to be defeated at once by the body forces leaving relative immunity.

3. Sensitiveness to tuberculin waxes and wanes through different periods of life.

Out of twenty-one medical students in London in 1928 I found eight negative to Von Pirquet's test.

Personally, I think the intracutaneous method more reliable, but the Von Pirquet method, or some variation of it, is used by many workers. Dr. Gunter (5) uses preparations of Tuberculin Albumose Frei (T.A.F.), and makes dilutions of 1 in 10, 1 in 100 and 1 in 500 ; he places a drop of each solution and a drop of normal saline as control on the fore-arm and makes a slight scratch over them. He writes : " A reaction with 1 in 10 tuberculin or stronger denotes in-activity, a reaction of 1 in 100 activity and 1 in 500 great activity. The degree of activity does not, of course, signify the seriousness of the condition of the moment, but it implies that active processes are going on, and whilst this is the case there is every probability of the disease progressing, unless steps can be taken to bring about arrest." Whilst agreeing in general with these views, I prefer to regard the test as evidence of infection only and to judge of activity by other signs and symptoms.

A third method of using tuberculin as a test is by sub-cutaneous injection. 0·2 c.cm. of dilution 4 (0·2 mg.) O.T. is

injected subcutaneously, and if there is no reaction three days later 1 c.cm. of dilution 4 (1 mg.) is injected. If no reaction follows this after three days 0·5 c.cm. of dilution 3 (5 mg.) is injected. A positive result produces :—

1. A local reaction consisting of redness and swelling at the site of the injection.

2. A general reaction consisting of malaise and a temperature which may rise to 102° F. or more.

3. A focal reaction which consists of inflammation round the various tuberculous lesions.

In the case of the lupus this focal reaction can be seen ; in pulmonary tuberculosis it may be indicated by X-ray evidence, increased crepitations, more sputum and, possibly, some hæmoptysis. A focal reaction is rare, and even when it does occur it is not often serious ; indeed, when it subsides it may be followed by actual improvement. In my opinion, the danger of this test, and in fact of tuberculin treatment, has been much exaggerated, but I have seen disastrous results follow a severe tuberculin reaction, and do not, therefore, advocate this method of testing, especially as the intracutaneous method gives equally reliable results.

Lastly, Moro's method of applying the tuberculin test may be mentioned. This consists of rubbing into the skin an ointment containing equal parts of lanolin and O.T. If positive, redness appears over the surface. This method may be used for infants when objection is made to an injection or even to scratching the skin, but failing the intracutaneous method I should always advocate Von Pirquet's test.

Complement Fixation Test

The Wassermann test for syphilis proved so satisfactory that it was hoped to apply similar methods in the diagnosis of tuberculosis. In practice, however, the usual difficulty arose, namely, how to distinguish active from non-active infection. Moreover, a positive result not infrequently is obtained in those who are apparently well and remain so

whereas a negative result is sometimes obtained in patients with definite and active tuberculosis. Alcock, Douglas and Lucey (6) obtained a positive result in 90·6 per cent. T.B. positive cases, but it was also positive in 36·5 per cent. T.B. negative cases. In arrested cases who had at some time been T.B. positive the test was positive in 47 per cent. only.

A positive complement fixation test may be taken as evidence in favour of the patient having or having had tuberculosis and a negative one as evidence to the contrary. Opinion as to the weight of this evidence varies enormously amongst different workers, but the majority do not regard it as of any special value except as giving some additional evidence one way or the other in a doubtful case.

Sedimentation Test

I shall refer to this test when dealing with prognosis, and mention it here only to say that it has but little value in diagnosis. It is true that in the majority of cases of pulmonary tuberculosis the sedimentation rate is increased, but so it is in many other conditions, and the doubtful case of pulmonary tuberculosis where there were no toxic symptoms and a fair condition of general health would quite likely show a normal sedimentation rate. Dr. Heaf (7), after testing 150 patients at King Edward VII. Sanatorium, Warwickshire, writes : " We wish to emphasise that the test is useless for diagnostic purposes."

Arneth Count

Arneth attached importance to the indentations or number of individual nuclei in the polymorphonuclear neutrophile leucocytes. He found in healthy individuals :—

Mononuclear forms with round or indented nucleus, 5 per cent.

Forms with two nuclei, 35 per cent.

Forms with three nuclei, 41 per cent.

Forms with four nuclei, 17 per cent.
Forms with five nuclei, 2 per cent.

In tuberculosis he found a predominance in the forms with one or with two nuclei. In one fatal case of pulmonary tuberculosis he found 46 per cent. with one and 49 per cent. with two nuclei. Professor Cummins (8) discusses the value of this test and the sedimentation test, and regards them of value as additional evidence.

Vital Capacity

As with the sedimentation rate, vital capacity is altered by many conditions besides tuberculosis and is of more value in estimating prognosis and the result of treatment than in aiding diagnosis. At the same time a normal vital capacity does undoubtedly render a diagnosis of active pulmonary tuberculosis very unlikely.

System of Diagnosis

At the Trudeau Sanatorium, Saranac Lake, New York State, stress is put on certain signs and symptoms which are considered of special importance, these are :—

Tubercle bacilli in the sputum.
Persistent râles in the upper part of the chest.
Parenchymatous lesion as shown by X-ray.
Focal reaction as determined by X-ray after subcutaneous test of 10 mg. or less of O.T.
History of unexplained hæmoptysis.
History of unexplained pleural effusion.

In addition to this, symptoms of toxæmia, such as fatigue, loss of weight or strength, fever over 99° F. in men or 99·6° F. in women, pulse over 90 in men or 96 in women, are regarded as indicating activity if not explained on other grounds.

A positive complement fixation test is not considered to indicate pulmonary tuberculosis, nor is a reaction to 10 mg. O.T. subcutaneously, provided there is no focal reaction as shown by X-ray or increased râles heard on auscultation.

At Trudeau, patients sent for diagnosis of pulmonary disease, are classified under four headings :—

Non-tuberculous. No direct evidence.

Suspected tuberculosis. A history of unexplained pleural effusion or unexplained hæmoptysis, but not both. No other evidence of a lung lesion.

Non-clinical pulmonary tuberculosis. Here X-ray shows a parenchymatous lesion not explained by other causes, but there are no symptoms and no tubercle bacilli have ever been found in the sputum.

Clinical tuberculosis. In this group there are, or have been, definite symptoms, together with one of the first four cardinal signs already mentioned or both unexplained hæmoptysis and unexplained pleural effusion.

This may be taken as a very good system of diagnosis. It is important to remember that there are two problems to be decided in diagnosis.

1. Is the patient infected with tuberculosis ? and if so,
2. Are the symptoms (or some of them) due to the tuberculosis or to some other cause ?

REFERENCES

(1) Fowler, Sir J. Kingston. "Pulmonary Tuberculosis," p. 172. London, 1921.

(2) Elliott, J. H. *Tubercle*, 1925, **VII.**, p. 122.

(3) Krause, Allen K. *Am. Rev. Tub.*, **XVIII.**, p. 60.

(4) Heimbeck, J. "Immunity in Tuberculosis," *Auch. Int. Med.*, 1928, **XLI.**, p. 336.

(5) Gunter, F. E. "Tuberculosis in Practice," p. 58. London, 1928.

(6) Broughton-Alcock, W., Mackenzie, Douglas and Lucey, H. C. *Lancet*, 1925, *i.*, 1331.

(7) Heaf, F. R. G. *Tubercle*, 1926, **VIII.**, p. 97.

(8) Cummins, S., Lyle and Acland, C. M. *Tubercle*, 1927, **IX.**, p. 1.

CHAPTER IV

RADIOLOGY IN PULMONARY TUBERCULOSIS

The great value of radiology in pulmonary tuberculosis is now generally admitted ; indeed, the modern tendency is to overrate rather than underrate its importance. In order to obtain full value from X-rays the work should be done by a competent radiologist, and in chest cases especially considerable experience in interpreting the shadows is required.

Dr. Melville (1) thinks the clinical and radiological examinations should be undertaken by separate individuals for the following reasons :—

1. Whichever is done first, the mental balance must be influenced.

2. The patient gets the benefit of two individual examinations and opinions.

3. The physician who becomes his own radiologist must lose that fineness of outlook and that power of summing up all the evidence which are so important.

Of course, in gross disease the condition is obvious, both clinically and radiologically, nor is it necessary to trouble the radiologist when the X-ray examination is intended to show some simple change, such as the degree of lung collapse before or after a pneumothorax refill, but all important or doubtful radiological work should be done only by the expert radiologist. For example, before thoracoplasty, where it is of the utmost importance to know the condition of the better lung, for on this the whole success of the operation and the life of the patient depends, the opinion of a radiologist on the film and screening of the chest is essential.

Before studying the X-ray shadows of an abnormal chest

it is necessary to understand those of a normal one, or rather of a healthy one, for certain abnormalities which produce shadows are consistent with perfect health. Dr. Riviere (3) discusses the X-ray appearances of the healthy chest and points out that in infancy there is little to see in the normal lung field outside the root shadow, but as time passes trunks and twigs of the lung network become visible, and at school ages may be very striking, especially as regards strands passing out fanshape from the root. These appearances are certainly compatible with health, and in Dr. Riviere's experience often become less obvious when adult life is reached. These shadows may be due to catarrhal attacks, infective fevers or fleeting tuberculous changes, but they certainly do not indicate active or clinical tuberculosis.

In the adult just outside the central shadow and partly covered by the heart on the left side can be seen shadows of the lung roots, and these are due to the main bronchi, blood vessels and lymphatics. Running out from these central root shadows are striæ passing into the lung tissue and composed of bronchi and their accompanying vessels. The bronchi appear as double shadows and look like railway lines and the blood vessels as streaks resembling the branches of a tree. If seen in optical section, the bronchi may appear as vague rings or small cavities and the blood vessels as dense nodules which may be mistaken for calcareous glands if in the section there happen to be much crossing and recrossing of the vessels.

It is important to recognise the round breast shadow in the healthy chest; it may be very prominent, and I have known it to be mistaken for a tumour.

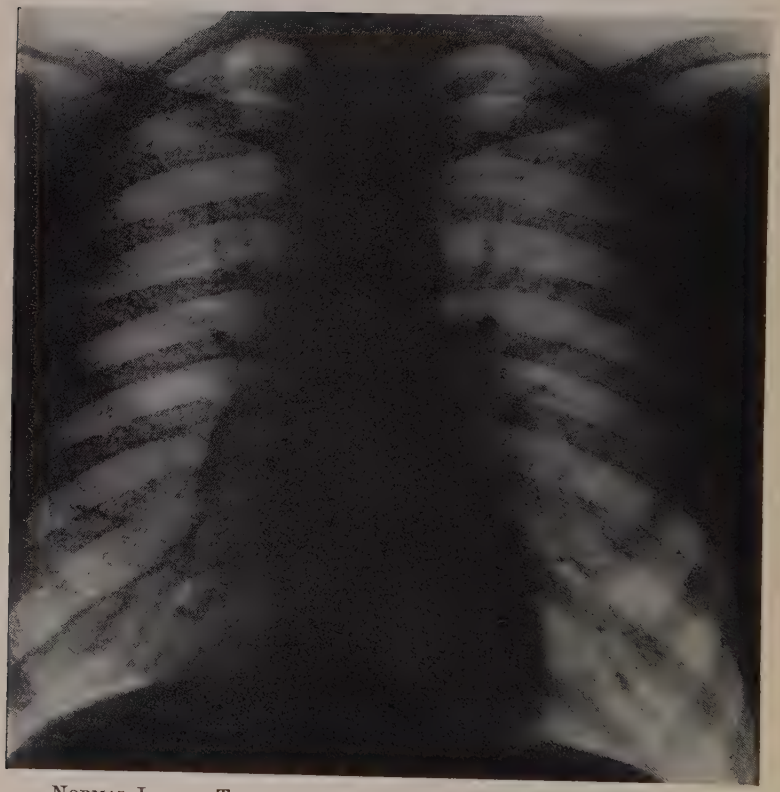
One must also remember that the lung forms a cupola fitting over the dome of the diaphragm, so that the dense shadow of the diaphragm does not indicate the lowest level of the lung, for there is lung tissue below this around the dome of the diaphragm.

PLATE I

Normal Lung.

[*To face p.* 34

PLATE II



Normal Lung. The heavy root shadows in this case have no pathological significance.

Dr. Lynham (4) finds it more frequent for the radiological report to indicate disease when the physician finds no signs than for the opposite to occur, and in my experience X-ray findings usually indicate more disease than would be expected from physical examination. As the result of post-mortem examination shows that there is almost invariably more disease present than the physical signs indicated, one must agree that X-rays can demonstrate the presence of disease when there are no physical signs. It sometimes happens that physical signs are present and tubercle bacilli exist in the sputum, and yet no definite X-ray abnormalities are noted. Such cases are rare, but they go to show that X-rays should be combined with other methods in the diagnosis of a difficult case and should not be relied on alone. Dr. Melville (1) says :—

1. Definite infiltration can be demonstrated upon an X-ray film at quite an early stage of the disease, and frequently before definite physical signs are evident.

2. X-ray evidence extending over a period of some months and consistently negative may be taken as conclusive evidence of the non-existence of pulmonary tuberculosis.

3. That in cases in which physical signs are present it may be assumed that the initial stage has passed, and in such cases the X-ray picture will show, as a rule, much more extensive disease than can be demonstrated by physical examination.

One common source of failure to interpret X-ray findings is the attempt to draw conclusions from a film alone, and often a poor film. In chest work it is essential to screen the patient, as this will show the movements of the chest and diaphragm, hypertranslucency or failure of a certain area to light up normally during breathing, movements of heart and mediastinum during inspiration and expiration, etc., and one can, moreover, move the patient so that his chest can be examined from several angles. A film will often show detail which was overlooked on the screen, but no

X-ray examination of the chest is complete without careful observation under the screen.

Another error, common amongst those who have but little experience of chest work, is to centre the tube incorrectly and to have the patient in a faulty position, so that the shadows are distorted and misleading. There may be so much error that the heart appears on the right side of the chest in a normal case.

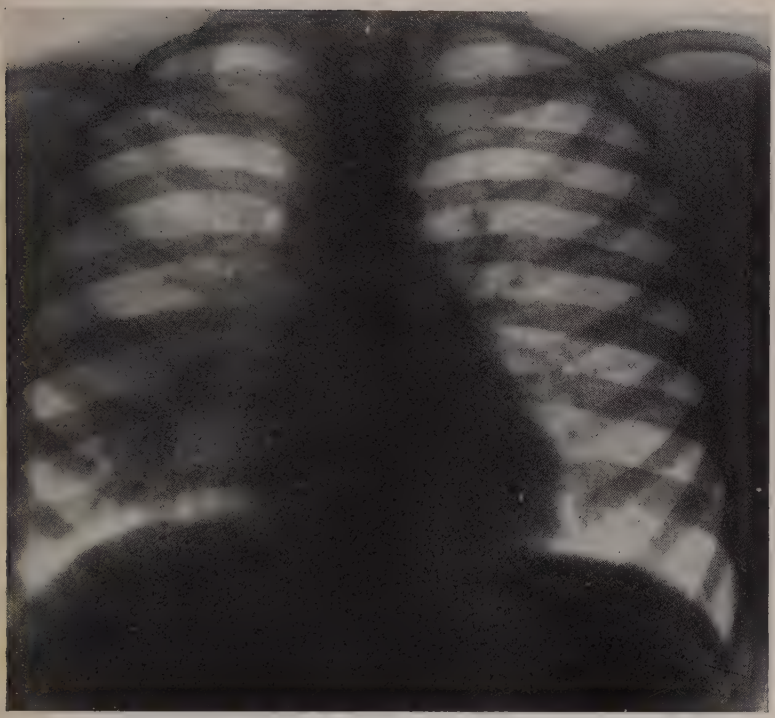
The tube should be at least 3 feet from the patient. Some like it to be 6 feet, as this gives a beam that is almost parallel and a clearer picture, but requires much more power or a long exposure.

There are two common heresies referred to by Dr. Melville (1). One that of so-called peribronchial tuberculosis. In certain cases, and especially those who have lived long in a city or suffered from chronic bronchitis, dense striæ are seen radiating into the lung tissue from the hilum, and when these were very dense they were supposed to indicate tuberculous spread along the lymphatics accompanying the bronchi, and hence the term peribronchial phthisis arose. They are but an exaggeration of the normal linear markings, and the term peribronchial phthisis is not now used by radiologists, nor is such a condition known in the post-mortem room.

The other heresy is hilum tuberculosis. That enlarged bronchial glands may exist and be seen by X-ray, and that they may be of tuberculous origin is not denied. But that X-ray examination can detect tuberculosis of the mediastinal glands before the disease has spread into the lung tissue is another matter. The infection passes from the lung to the glands, and, although a tuberculous mediastinal gland may suppurate into lung tissue and set up acute disease, in the ordinary way the infection does not pass against the lymphatic stream from the gland back into the lungs.

On this subject Dr. Lynham (4) writes: "I have seen many cases with root areas involved, but they all show lung tissues involved as well."

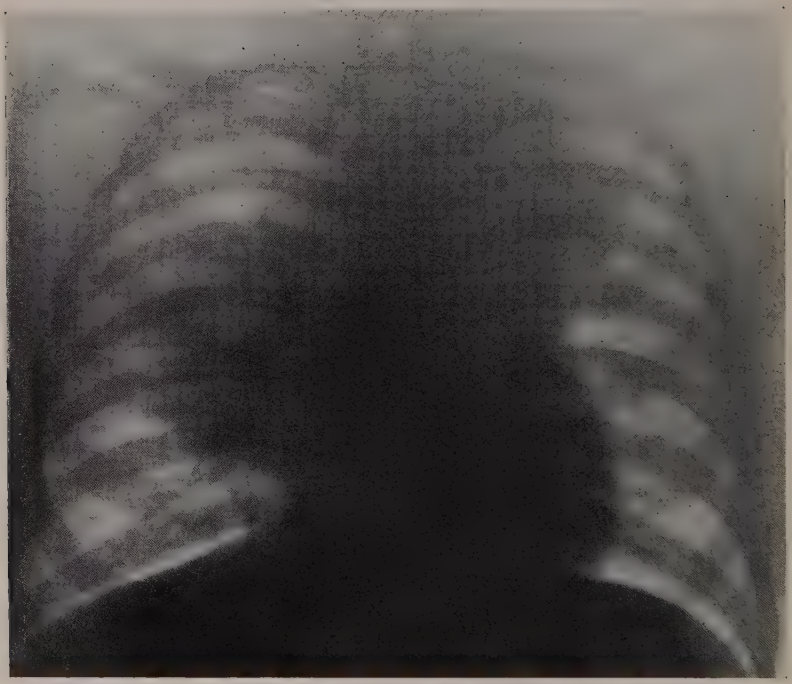
PLATE III



NON-TUBERCULOUS PNEUMONIA IN A CHILD. THE SHADOW PERSISTED FOR MANY MONTHS, BUT THE CONDITION EVENTUALLY CLEARED UP COMPLETELY.

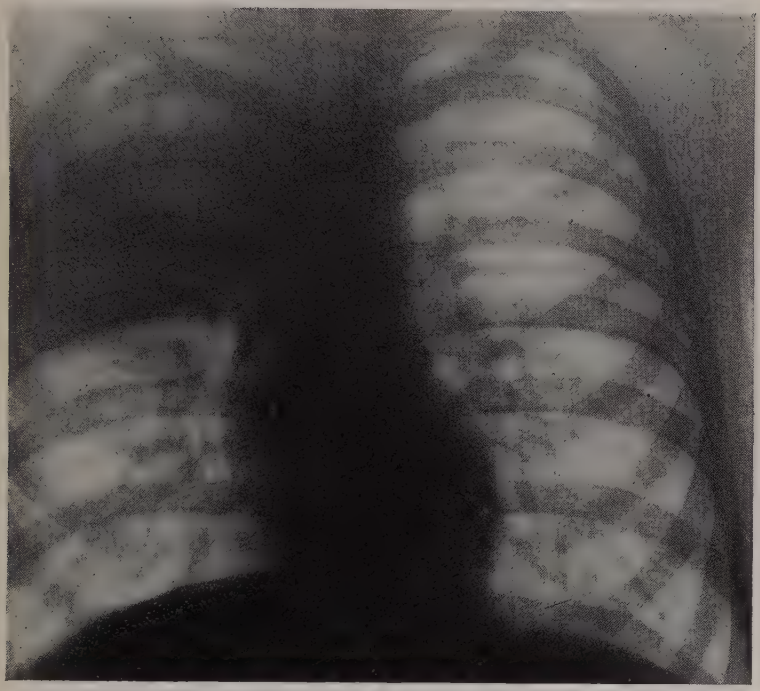
[*To face p.* 36.

PLATE IV



Lymphadenoma. Great Enlargement of the Glands. Compare with Plate III, where the shadow on the right is due to pneumonic lung.

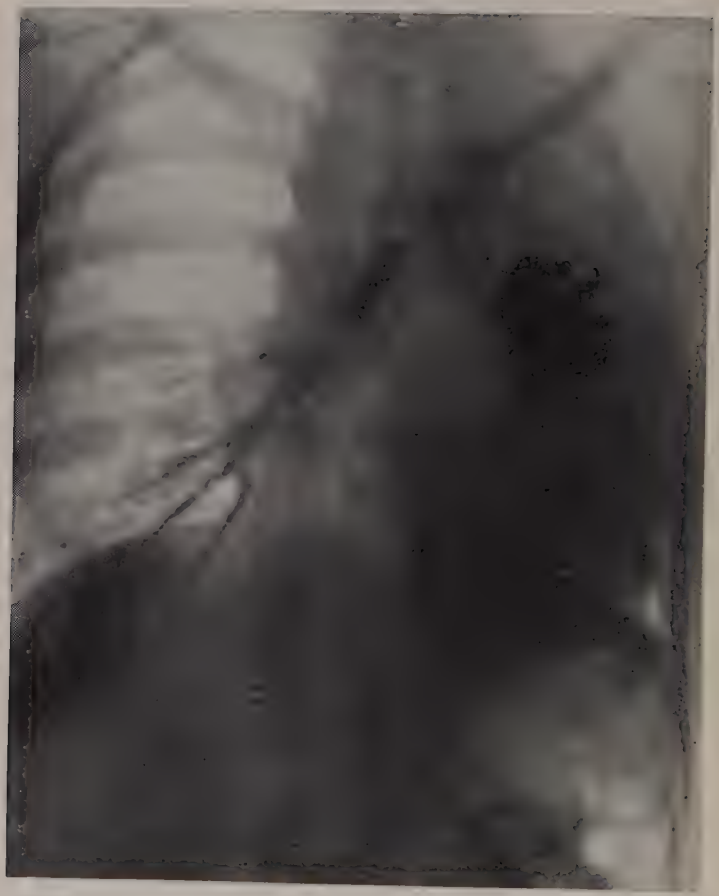
PLATE V



Non-tuberculous Apical Pneumonia in a Child.

[*To face p.* 36.

PLATE VI



TUBERCULOUS FIBROSIS WITH A LARGE CAVITY CONTAINING LIPIODOL. NOTE THAT THE TRACHEA AND MEDIASTINUM ARE DISPLACED TO THE LEFT.

One must also remember that considerable exaggeration of the hilum shadows may exist without any enlargement of the glands. Dr. Lynham says that when screening, the patient should be made to inspire and then expire to his fullest capacity. It will then be found that " solid bodies, such as calcified glands, retain their shape ; fibrotic areas show up at full inspiration like the fine branches of a moving tree ; caseating areas resemble crows' nests in tree branches ; whilst fluid areas, such as the large veins, undergo a change of shape which one can only describe as amœbic."

It is largely because of the difficulties in technique, in the interpretation of shadows seen, and in the art of screening that X-ray examination does not always receive the credit which it deserves, but its value is being more and more recognised by those who see many cases of thoracic disease and base their judgment on the reports of skilled radiologists.

Apart from being almost indispensable in controlling artificial pneumothorax treatment, and in judging the effects of such operations as thoracoplasty or phrenic evulsion, X-ray examination in relation to pulmonary tuberculosis is of special importance for two purposes :—

1. Differential or early diagnosis. As an example of differential diagnosis I may mention the case of a woman who had been in a sanatorium for many months with a diagnosis of pulmonary tuberculosis based on repeated hæmoptyses. After injecting lipiodol into the trachea X-ray examination revealed complete blocking of one of the larger bronchi. This was found to be due to a fibroma, which was subsequently removed by operation, and the patient recovered.

With regard to early diagnosis, X-ray examination will not show the very earliest changes due to tuberculosis of the lung, but it will almost invariably reveal disease before the appearance of physical signs. Dr. Melville (2) describes nine cases where X-ray evidence of pulmonary tuberculosis were

present and the disease correctly diagnosed, but in which no physical signs were found after careful examination by competent physicians.

2. The other great function of X-ray examination is to assist in forming an opinion as to the extent and type of disease in cases where treatment depends on these matters.

For example, before thoracoplasty is performed it is essential to know that there is no active disease in the better lung, and also that the patient has powers of resistance, as evidenced by the presence of fibrosis.

REFERENCES

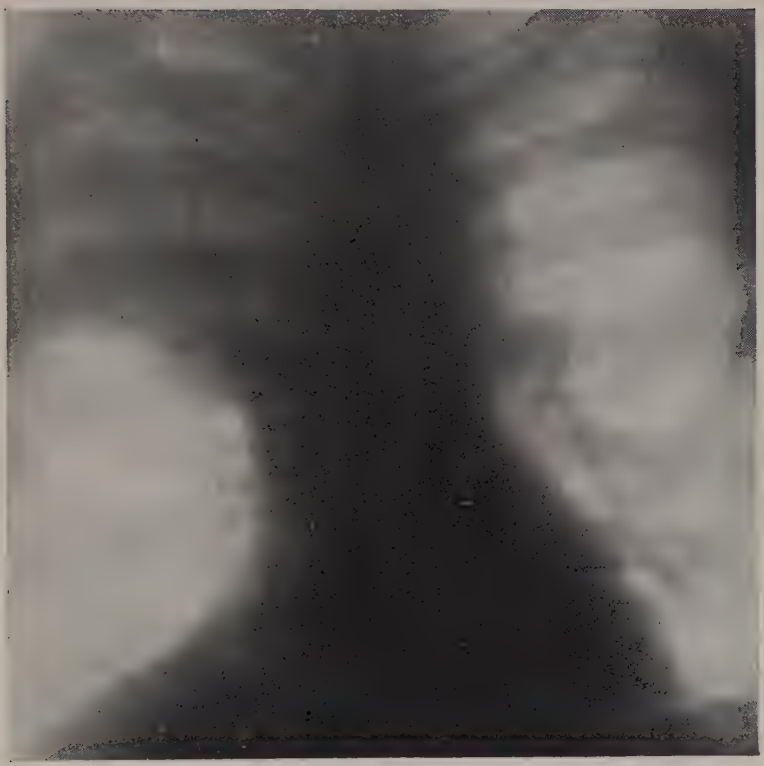
(1) Melville, Stanley. *Post-Grad. Med. Journ.*, 1929, **IV.**, 79.

(2) Melville, Stanley. *Proc. R. S. Med.*, 1923, **XVI.**, (Electro-therap. Sect.), p. 31.

(3) Riviere, Clive. *Tubercle*, 1925, **VI.**, 382.

(4) Lynham, J. E. A. *Tubercle*, 1925, **VI.**, 473.

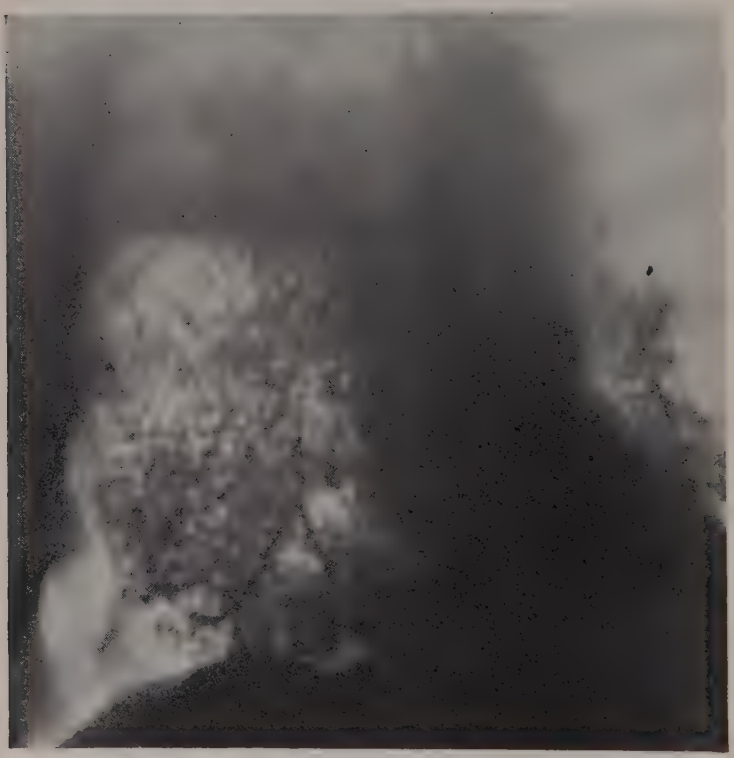
PLATE VII



Carcinoma of Bronchus which has led to Collapse of the Upper Part of the Right Lung.

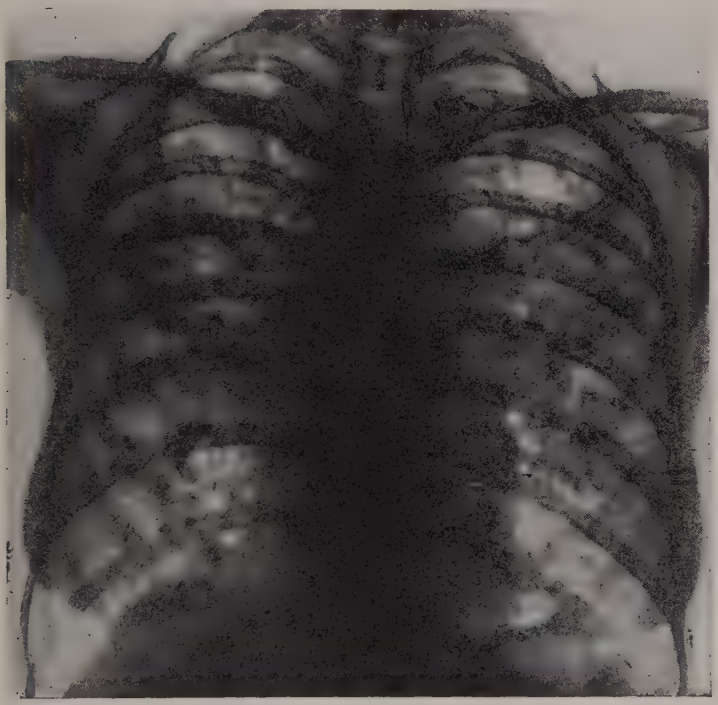
[*To face p.* 38.

PLATE VIII



The same Case as Plate VII after Lipiodol has been injected and the Lung partially collapsed for Diagnostic Purposes. Note that no lipiodol has entered the upper part supplied by the affected bronchus.

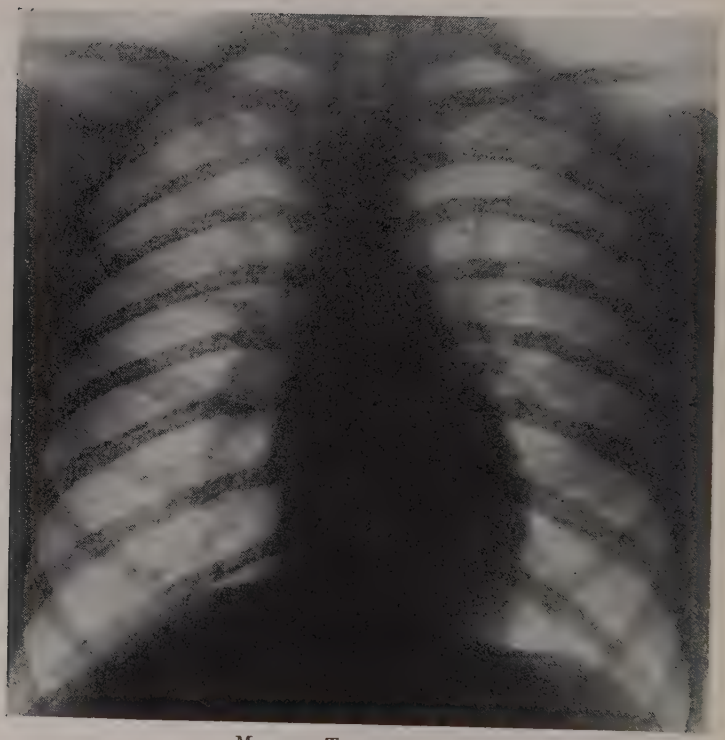
PLATE IX



TUBERCULOUS BRONCHO-PNEUMONIA.

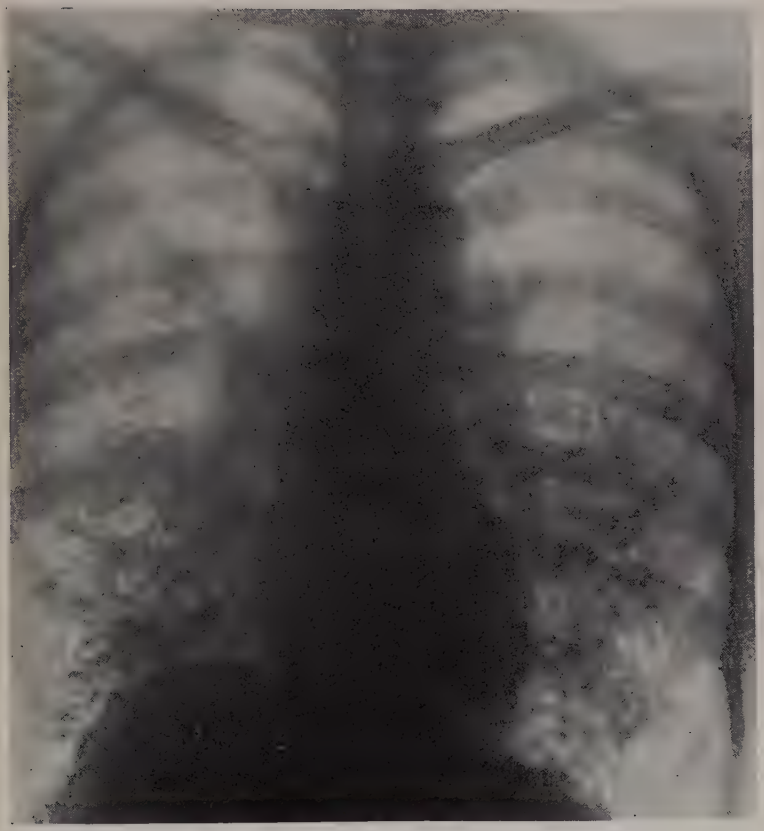
[*To face p.* 38.

PLATE X



Miliary Tuberculosis.

PLATE XI



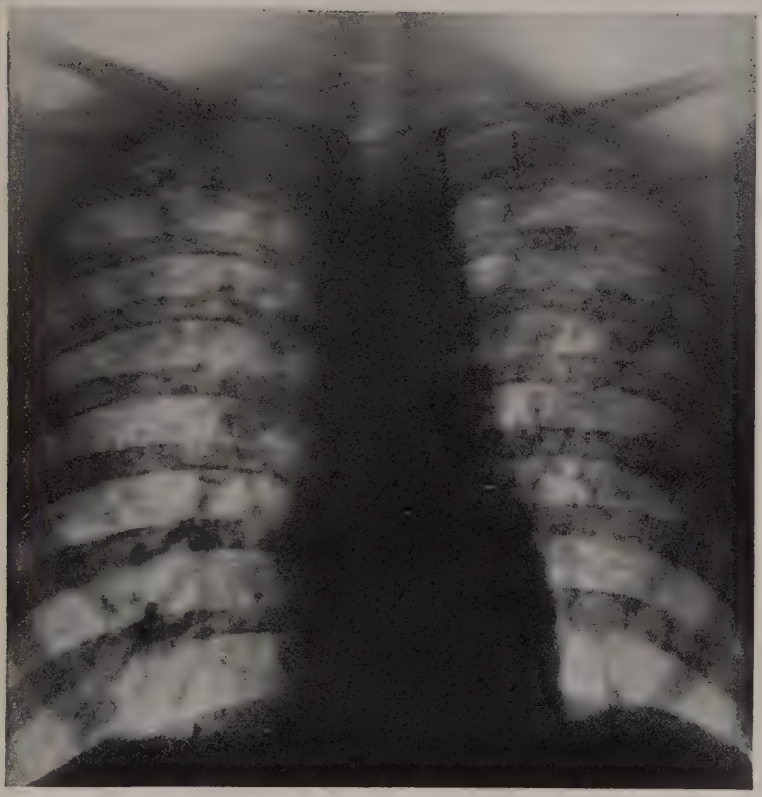
CARCINOMATOSIS.

[*To face p.* 38.

PLATE XII

Silicosis.

PLATE XIII



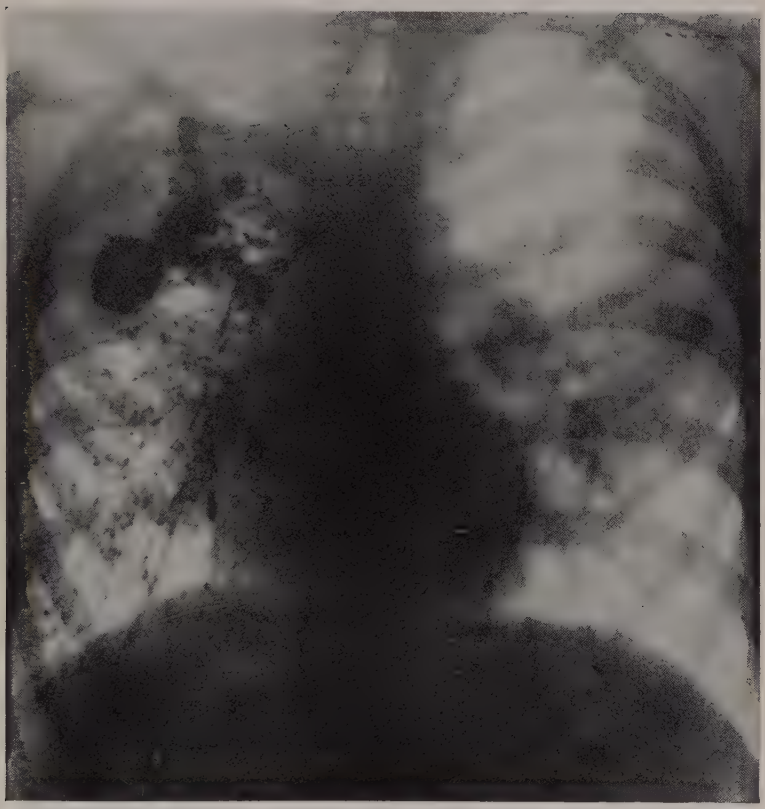
Chronic Tuberculous Infiltration.

[*To face p.* 38.

PLATE XIV

Extensive Tuberculous Cavities after Lipiodol has been injected.

PLATE XV



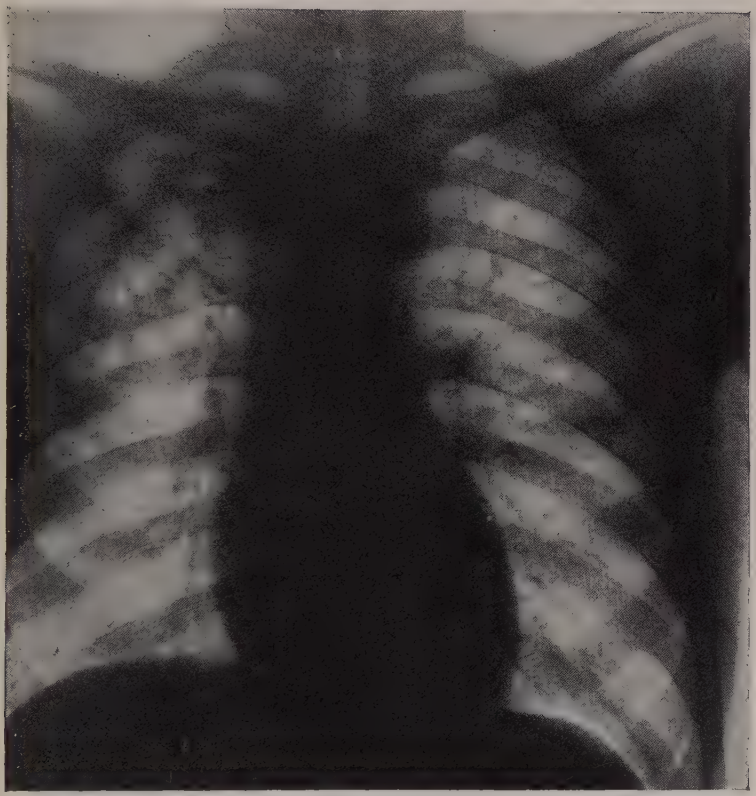
TUBERCULOUS CAVITY. LIPIODOL HAS BEEN INJECTED, AND THE PATIENT IS IN THE RECUMBENT POSITION.

[*To face p.* 38.

PLATE XVI

Same Case as Plate XV with the Patient in the Exact Position. Note the fluid level of the lipiodol.

PLATE XVII



Chronic Fibroid Tuberculosis of Right Apex before the Injection of Lipiodol.

[*To face p.* 38.

PLATE XVIII

THE SAME CASE AS PLATE XVII AFTER LIPIODOL HAS BEEN INJECTED. NOTE THE LARGE CAVITY.

CHAPTER V

PROGNOSIS

In no disease is the prognosis so uncertain as in pulmonary tuberculosis, and it is quite impossible to foretell the future of any given case. At the same time if a large number of cases are taken it is possible to give a correct prognosis in a big percentage.

Physique.—Patients who are physically weak, and especially those who have badly developed or deformed chests, tend to do badly if they become tuberculous. Insurance statistics show an increasing mortality from pulmonary tuberculosis as the weight of the individual falls below normal.

Temperament.—The temperament of the patient is also of the greatest importance. Kingston Fowler (2, p. 184) wrote: "No fool ever gets rid of tuberculosis of the lungs. He may be no fool in relation to literature, science or art, but if he is so in relation to his own well-being he is doomed for certain." The patient with a placid temperament who takes things as they come and does not look too far ahead has the best chance of recovery. During the active stage he will follow out the treatment for his own sake, and, having reached the highest level of improvement, he will adjust his life so as to get the most out of it, in spite of any limitation of activity which may be necessary. Many people in all walks of life have done much useful work and left great names behind them in spite of having suffered for years from chronic pulmonary tuberculosis, and probably in many cases their lives were prolonged by the fact that they did work. It is play, far more than work, that kills in tuberculosis. If, therefore, one finds a patient steadily

working under suitable conditions and not worrying about his health the prognosis is always hopeful.

It often happens that a patient with a temperament that is bad for a tuberculous subject is able to control himself, and eventually becomes reconciled to his condition, but many patients are never able to change, and for them the prognosis is very grave.

There are three types which are especially bad :—

1. The patient who finds the necessary restrictions so extremely irksome that he has to disregard them in order to prevent a mental breakdown. The same applies to the patient who has not sufficient self-control to regulate his life to suit his state of health.

2. The patient who is over-nervous and unable to interest himself in anything except his own health. This class of patient is continually taking his temperature and is in a constant state of anxiety over the slightest ache or symptom that may arise. He will not take up any employment even when well enough to do so, and very rarely lives long, in spite of the great amount of trouble and thought he takes of his own health.

3. The opposite type of patient, who cannot realise that there is any need to take precautions unless he has some definite symptom, such as hæmoptysis, is also unlikely to do well. When he is febrile and feeling ill he will consent to go to a sanatorium and do his best to get well, but on leaving the sanatorium, if he has no symptoms and feels well, he will play games and return to his ordinary life, not from lack of self-control, but because he thinks he is cured. An attack of hæmoptysis or pleurisy will again lead him to seek advice, but as soon as he feels well he once more returns to a life of full activity. He is quite willing to receive treatment when he has a breakdown, but does not see the necessity of taking any steps to prevent one.

As an example of this type of patient the following case may be mentioned. A patient, aged thirty-two, who had

always been healthy, lived an active and athletic life and weighed 11 st. 5 lb. In April, 1928, he caught a cold, which was followed by a cough, sputum developed and tubercle bacilli were found. On examination occasional crepitations after cough were heard at the right apex, but there were no other signs. X-ray showed a slight shadow at the right apex. He was afebrile and felt well in himself. He was strongly advised to have a period of rest, followed by sanatorium training, and the prognosis was considered good. However, he refused to rest or go into an institution, saying that he was not ill enough for sanatorium treatment, and felt sure that he would get well after a short open-air holiday with plenty of milk and good food. He took a holiday, playing golf and living what he thought an ideal life, but in May he had lost considerable amount of weight and began to feel ill. In June he had extensive signs in the upper part of the right lung, crepitations being heard as low as the angle of the scapula behind. At the root of the left lung crepitations were heard also, and X-ray showed great extension in the right lung and shadows in the upper part of the left. He rapidly got worse and died in December. If he had had a large hæmoptysis at the beginning of the illness it would probably have saved his life, as he would have treated the condition seriously from the onset and had treatment in the early stages of the disease.

Environment.—It is necessary, of course, to distinguish between a bad temperament and inability to have the necessary treatment, or to lead the correct life owing to financial or other reasons. The patient whose home conditions are good, and who can afford a reasonable amount of comfort, has a far better chance than one who has to return from a sanatorium to poverty, unsuitable work and bad home conditions. In judging the prognosis the greatest weight must be given to the sort of life the patient will have to lead after his treatment is finished.

Extent of Disease.—The extent of the disease is also

important, and it has been found that patients in Group II. of the Turban-Gerhardt classification (which is based on the anatomical extent of the disease) have a greater expectation of life than those in Group III, but less than those in Group I. Other things being equal, therefore, the greater the amount of disease the worse the prognosis. The type of disease is, however, of more importance, for if there is evidence of resistance, as indicated by fibrosis and even by cavities, the outlook is much better than in the case of a caseating lesion which spreads rapidly without any sign of arrest. It is quite a mistake to regard a cavity as indicating a bad prognosis ; it is the result of Nature's method of cure.

Response to Treatment.—The response of the patient to treatment is a very valuable guide to prognosis. In the great majority of cases a patient improves when he first comes under treatment, and this is probably due to the rest, which is the basis of almost every form of treatment in the initial stages, but after a few weeks some patients fail to improve or begin to lose ground, and for these the outlook is much less hopeful than for the patient who steadily improves, and is able to go from stage to stage of the treatment without a setback.

Age.—The type of disease varies to a considerable extent according to age. In young children tuberculosis tends to be arrested or fixed in the glands ; if it occurs in the lungs it is frequently part of a general infection and the outlook is hopeless. The prognosis in the case of glandular tuberculosis in children is good. Delicate children who are undergrown, and perhaps have a slight dry cough with some enlarged hilum shadows seen by X-ray, are often diagnosed as having tuberculous mediastinal glands, and no doubt some of them have, but they are no more likely to develop tuberculosis of the lungs than other children. It is, of course, important to improve their general state of health, and a prolonged stay at the seaside with healthy exercise and outdoor games is very necessary whether the debility is really due to tubercu-

lous glands or not, but it is wrong to class them as "pre-tuberculous" in the sense that they are liable to become consumptive.

About the age of puberty pulmonary tuberculosis usually runs a very acute course, and this is the age of greatest mortality. The prognosis, therefore, is very grave, although recovery may take place even in cases which appear to have very extensive and rapidly-spreading disease. The prognosis here depends to a great extent on the response of the patient to treatment. In adult life the disease is generally chronic, and arrest takes place under proper treatment. In old age pulmonary tuberculosis is not common, but when it does occur the prognosis is serious.

Family History.—This is usually of no great importance in prognosis. The fact that an aunt or cousin, or even brother or sister, died of pulmonary tuberculosis does not make the outlook more serious. Indeed, I have found that patients who develop an acute form of tuberculosis of the lungs some few months after nursing or being in close contact with an infected relative, often respond readily to treatment and make good recoveries. In some families, however, there seems to be a special susceptibility or lack of resistance, and one must take a grave view when a member develops the disease. In one family the mother died of pulmonary tuberculosis when thirty-three, two of her sisters died of it between twenty and thirty. Her son died of acute tuberculosis when nineteen, and her daughter contracted it at the age of twenty-three. Although her general condition was good and the signs very few when she was first seen, one had to take the family history into account in giving a prognosis. The disease did run an acute course and she lived less than a year.

I once saw a woman of sixty-five with signs of early pulmonary tuberculosis, but the disease ran a rapid course and she died in six months. A few weeks before she became ill she had been nursing her sister, who died of the same

complaint. Although there was no history of tuberculosis in her father's or mother's family, she had lost two brothers and four sisters from pulmonary tuberculosis. Their ages at death were thirty, forty-four, seventy-six, seventy-six, seventy-eight and eighty-one.

Drolet (6) records an investigation into the effect of heredity on tuberculosis. He came to the opinion that tuberculous parents confer some degree of immunity on their offspring which renders them actually less liable to contract the disease than are the children of healthy parents. His results seem to show that children do not inherit any susceptibility to tuberculosis. The matter is affected by many factors, such as contact with infection, home conditions, other illnesses, etc. Moreover, the difficulty of following up all members of the family throughout the whole of their lives and then getting reliable evidence of death is so great that figures are apt to be most misleading. Family histories where several members develop tuberculosis at about the same age are not uncommon, and frequently the same type of disease attacks each member, and it runs a similar course in each case.

It is difficult to explain such cases without supposing that there is some inherited quality which renders them susceptible. Of course, children brought up in a home with tuberculous parents are sure to be infected, and apart from any inherited predisposition, repeated mild infections might confer some immunity and large infections would produce actual disease.

Dr. Ford (7) studied the histories of over 1,000 cases of pulmonary tuberculosis and found that 79·5 per cent. gave no family history of tuberculosis. His results also suggested that a family history of tuberculosis did not increase the gravity of the prognosis. On the contrary he found that those cases whose mothers had been tuberculous did much better than the others.

Temperature.—Before discussing temperature it may be

as well to say a few words about the best method of taking it.

The chief difference of opinion concerns the oral and rectal methods. Probably the rectal method is slightly less liable to error than the oral, but it has certain disadvantages. Not only is it unpleasant for the patient or nurse, but it is undoubtedly apt to aggravate any nervous or hypochondriacal tendencies on the part of the patient. A patient who has been accustomed to having the temperature taken by the mouth finds himself in another institution where it is taken by the rectum. If neurotic he will immediately think that the slightest variation of temperature is of extreme importance, his spirits will rise and fall with his temperature, and long after he should have discarded his thermometer he will secretly use it, and a mysterious fit of depression may be accounted for by the fact that his rectal temperature was 99·4 after tea. I have frequently noticed, especially in foreign institutions, how common a topic for conversation is the temperature chart, and what a bad effect it has on the patients as a whole. It is quite true that a patient may worry about a temperature taken by the mouth, but it has not the same significance to most of them. The rectal method suggests that the slightest rise of temperature is so vital that the most accurate method of obtaining it must be employed and the natural result is that a slight elevation will cause the patient serious anxiety. I do not wish to underrate the value of the temperature chart in pulmonary tuberculosis. On the contrary, I entirely agree with Sir James Kingston Fowler that "the temperature is the guide to the activity of the disease," but I maintain that consideration for the mentality and temperament of the patient is of far greater importance than any slight difference between rectal and oral temperatures. For special purposes, such as after tuberculin or exercise, in diagnosis a rectal temperature may be useful, but in my opinion it is wrong to use it as a routine. I have often had the temperature taken by the

mouth, axilla and rectum simultaneously in order to demonstrate to my class how similar are the curves, and for

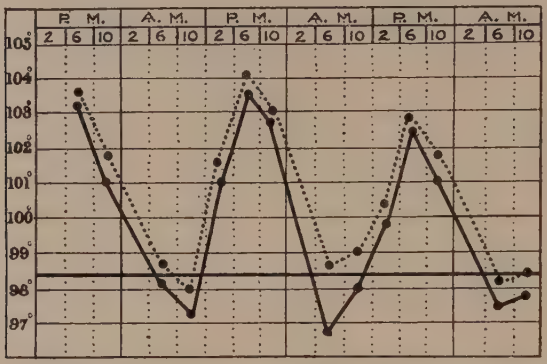


Fig. 1.—Dotted line, rectal temperature; solid line, mouth temperature.

the purpose of this book I had rectal and oral temperatures taken in three patients, and the results are shown in Figs. 1, 2 and 3. The higher curve is the rectal temperature,

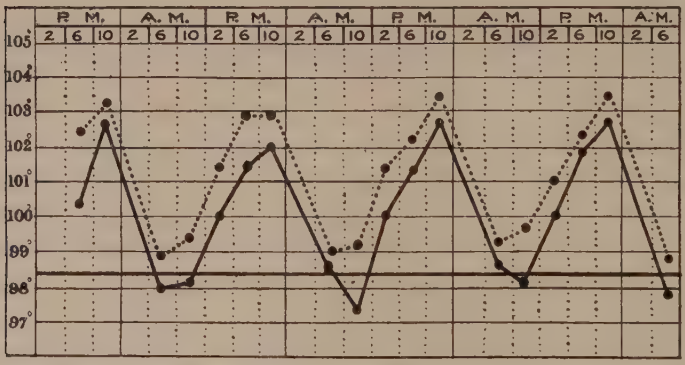


Fig. 2.—Dotted line, rectal temperature; solid line, mouth temperature.

but it will be found to rise and fall exactly as the oral one, and these charts support my contention that the objections to the rectal method outweigh its advantages.

With regard to the axillary temperature, this also corresponds with the oral and rectal method, but the axilla must be carefully dried first. It is unreliable to take the temperature in a stream of urine.

Dr. Colin Milne (1) writes: "By taking the oral and

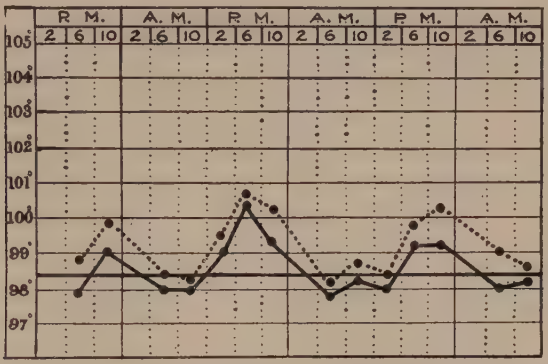


Fig. 3.—Dotted line, rectal temperature; solid line, mouth temperature.

rectal temperatures simultaneously, twice daily for three weeks in one series, I found the tracings on the charts to be almost parallel, the rectal being the higher by about half a

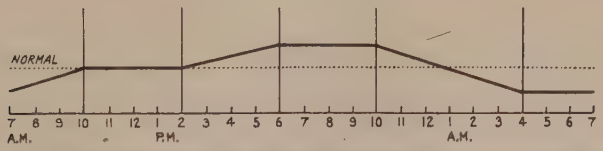


Fig. 4.—Diagram showing the daily variation of temperature in the normal subject.

degree." He thinks the oral method of taking the temperature is the most suitable.

The temperature in pulmonary tuberculosis may be taken as an exaggeration of the normal curve, being lowest about 4 a.m. and highest between 6 to 9 p.m. In the morning from about four to seven it remains low, and then gradually rises, reaching normal about ten. It remains there until two in the

afternoon, when it again rises until 6 p.m., and then reaches and remains at its maximum until 9 or 10 p.m., at which time the drop begins. The diagram on p. 47 illustrates the usual variation in the temperature, and it will be seen that by taking an early morning and evening temperature the minimum and maximum can be obtained.

When there is a big difference between the maximum and minimum the temperature is called hectic, and some have thought this is due to a superadded infection by pyogenic organisms. Koch thought a temperature of over 100·4 was due to the presence of septic organisms, and consequently should be taken as a contra-indication for tuberculin. It has been stated that various forms of treatment, climatic and other, should depend on whether the case is a pure tuberculosis, or complicated by mixed infection, and it has been claimed that the advent of mixed infection can be diagnosed by the temperature chart. Since it affects treatment and prognosis, as well as our views of the course of the disease, it is of great importance to consider how far, if at all, the temperature in tuberculosis is affected by secondary organisms. Of course, in long-standing cases of pulmonary tuberculosis, other organisms will collect, and tuberculous patients are just as liable as any to fever from other causes ; but there is no doubt that typical hectic fever is seen in cases of tuberculous peritonitis, pleurisy, etc., where post-mortem examination shows simply tuberculous disease. Sir James Kingston Fowler (2) says : " The parrot cry of mixed infection does not come from the pathologists." And again : " the term ' mixed infection ' has no place in relation to pulmonary tuberculosis, but of course other organisms are present in the lungs when breaking down of the lesions and the formation of cavities is in progress."

Thus we may consider the hectic type of temperature as an extreme exaggeration of the normal, and as indicating active breaking down of the lung tissue. The temperature is the guide to the activity of the disease, and all degrees

may be seen from the hectic in rapidly spreading tuberculosis to normal in the chronic fibrotic disease when there is little or no spread. It must not be forgotten, however, that there may be gradual spread of disease in a fibroid case without pyrexia, and, of course, in advanced cases where resistance fails the terminal stages are afebrile.

Apart from this usual type of fever, varying according to the acuteness of the infection, there are other types met with in tuberculosis. In the inverse type (Fig. 5) the morning temperature is high and the evening low, and it is of serious import. Sir James Kingston Fowler (2) writes that of 30 cases of tuberculosis of the lungs, mostly of the chronic variety, under his care in the Brompton Hospital, in which the inverse type of pyrexia was observed, 14 died in hospital, and 1 immediately after returning home; 8 left the hospital worse; 5 were relieved or about the same; 2 improved, but in these the inverse type of temperature was of only temporary duration, being present twice in one and eight times in the other.

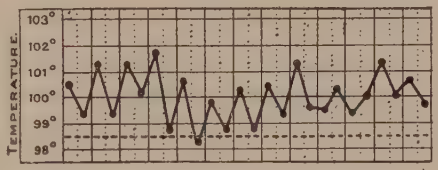


FIG. 5.—Showing inverse type of temperature.

The inverse type is usually seen in cases of remittent fever. The morning rise persists, but in the evening the patient fails to react, and consequently there is no rise of temperature. A high temperature in tuberculosis can usually be reduced by such drugs as cryogenin or pyramidon, but it rises again as soon as the drugs are stopped. In some cases of high remittent fever the drug reduces the evening rise of temperature, but has little or no effect on the morning one, so that an inverse type of temperature results, which takes on the usual form again as soon as the drug is discontinued. This is shown in Fig. 6.

The continuous type of temperature is often due to the

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occurrence of miliary tuberculosis, and is therefore a serious sign. If in a case of chronic pulmonary tuberculosis, or pleural effusion, the swinging temperature rises and the morning intermissions disappear, as shown in Fig. 7,

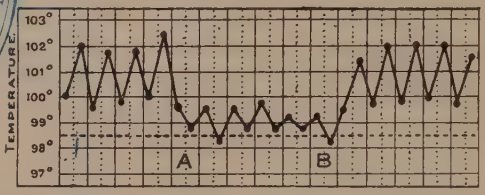


Fig. 6.—Remittent type of temperature. At A the patient was given cryogenin gr. v. twice daily and at B this was stopped.

miliary tuberculosis and a fatal termination must be expected.

The most serious temperature is when, in spite of much active disease, the patient becomes afebrile and at the same time shows signs of collapse. This indicates failure to react and complete loss of resistance, and, as seen in Fig. 8, is

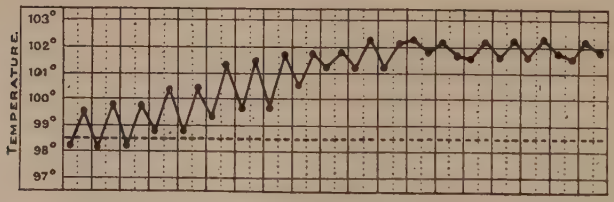


Fig. 7.—Intermittent type of temperature becoming continuous and high owing to the onset of miliary tuberculosis.

usually followed by a fatal issue in a few days, but a similar fall of temperature associated with improved condition of the patient would be a very good sign. Fig. 9 shows an intermittent temperature, becoming remittent as the disease progressed. At the onset the temperature was quite consistent with a favourable course, but as, in spite of rest and other treatment, it not only failed to subside but grew worse,

the prognosis became increasingly grave, and the patient died later with tuberculous broncho-pneumonia.

A persistent but slight rise of evening temperature from 99° to 99·4° F. is usually not due to pulmonary tuberculosis,

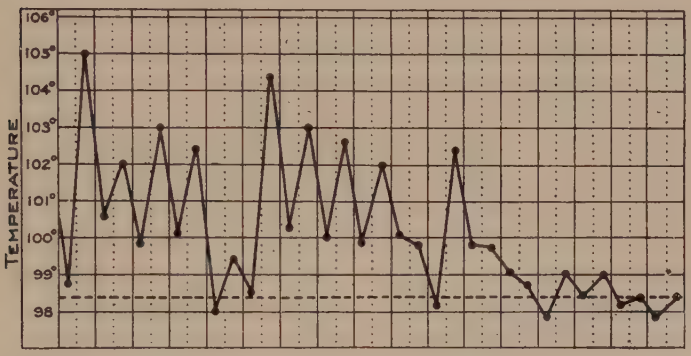


FIG. 8.—Fall of temperature owing to exhaustion of the patient.

but to some complication, such as tuberculous peritonitis or pleurisy. It may persist for many months, as in the following case: the patient, a man of twenty-three, developed pulmonary tuberculosis, and after a few months in a sanatorium

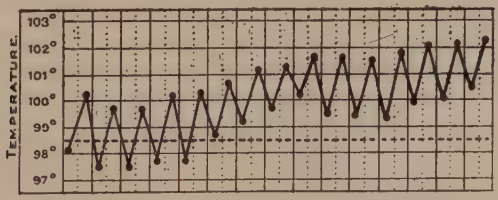


FIG. 9.—Intermittent type of temperature becoming remittent owing to increased activity of disease.

he improved, the signs in the chest showed that satisfactory fibrosis was taking place, tubercle bacilli disappeared from the sputum, and eventually the sputum dried up altogether. In spite of these favourable signs he continued to feel slack, and he always had an evening temperature of between

99° and 100° F., whether he was resting or up and about. He left the sanatorium and was living a quiet but fairly normal life at home, when suddenly, about eighteen months after the onset of his symptoms, he had an acute attack of vomiting and abdominal pain, which was thought to be due to food poisoning as it occurred immediately after a picnic. Five months later he had another attack, and laparotomy was performed. Extensive tuberculous peritonitis was found, and there were many adhesions, some of which were divided as they were obstructing the intestine. A period of rest following the operation restored him to health, and he no longer has the evening rise of temperature.

Lastly, there are two types of periodic fever in tuberculosis. One is seen in women and occurs as a slight premenstrual rise, beginning a week or ten days before each period and rapidly falling after the first or second day of menstruation. Such a slight variation in temperature is not peculiar to tuberculous patients, and is of no significance in prognosis, but occasionally there is a definite rise of temperature to 101° F. or 102° F. for a day or two before each period. This is very similar to a tuberculin reaction, and passes off when the period begins. These menstrual reactions are very rare, and are not of serious import. I have failed to prevent them by giving antituberculous serum.

A second type of periodic fever may occur every few weeks or months, and is usually associated with pocketing of sputum in a cavity or dilated bronchi. The patient coughs up a large quantity of sputum for a day or two, and then the temperature falls and the health improves for a time, when the same occurrence is repeated. Prognosis in these cases depends on the severity of the symptoms and the response of the patient to treatment, such as postural drainage, phrenic evulsion, etc.

The following conclusions may be drawn concerning the value of the temperature in prognosis :—

1. Pyrexia must be considered in conjunction with other

factors in regard to prognosis. For example, although a normal temperature is usually of good import, progressive fibrotic disease may occur without causing a rise of temperature and absence of temperature may be due to failure of resistance in the terminal stages of the disease.

2. The temperature is, however, the best guide to the activity of the disease. Other things being equal, the best prognosis should be given in cases who are afebrile in spite of exercise, and the worst in those who remain febrile in spite of rest in bed. An intermediate position is occupied by those who are afebrile whilst at rest, but febrile after exertion. The less exercise it takes to send up the temperature the more serious becomes the outlook.

3. The inverse type of temperature is of serious import.

4. When the type of temperature changes to a high continuous pyrexia the prognosis is very bad, for it indicates the occurrence of miliary or generalised tuberculosis.

5. A prolonged slight elevation of the temperature is usually due to some cause, such as tuberculous pleurisy or peritonitis.

6. Prognosis in cases of periodic rise of temperature depends on the cause of the rise and the response of the condition to treatment. Slight premenstrual rise of temperature is of no significance.

Sedimentation Test.—Although this test is now considered to be of little value in diagnosis, it is of undoubted use in prognosis and in determining the patient's response to treatment.

The test depends on observing the rate at which the erythrocytes fall, leaving clear serum above in a column of blood. There are many methods of performing the test. Some workers prefer to get the blood from a vein, but the following technique described by Drs. Trail and Stone (3) has the advantage of requiring only a few drops of blood from a pricked finger or ear :—

" To two volumes of a sterile solution of 3·8 per cent.

sodium citrate eight volumes of blood are added. The unit of volume is one drop of blood from a broad-ended glass pipette ; the blood is drawn from an ear or finger and mixed in a watch glass. A column of the diluted blood, 4 in. (*i.e.*, 100 mm.) long, is drawn by capillary attraction into a fine glass tube about 6 in. long and 2 mm. bore. The tube is sealed with sealing wax and placed vertically in a bed of plasticine. In this investigation the length of the clear fluid column above the sedimented red cells was measured at the end of an hour, in millimetres, this giving the result directly as a percentage. Any reading above 6 mm. for a man or 12 mm. for a woman is considered abnormal."

In a series of 360 patients at Midhurst Sanatorium, Drs. Trail and Stone found that of these 318 were clinically cases of pulmonary tuberculosis and 42 were not. Of the 318 an increased sedimentation rate was observed in 309 (97 per cent.), but of the 42 the rate was increased in only 13 (31 per cent.). They took a further series of 100 T.B. positive cases, who were under ordinary routine sanatorium treatment, and examined their sedimentation rate over a period of at least four months. The clinical condition of these patients on discharge fell into three groups :—

A. Improved or arrested with T.B. negative sputum. In this group the average range of sedimentation was from 23 on admission to 12 on discharge.

B. Improved but still T.B. positive. In this group the sedimentation rate ranged from 32 on admission to 22 on discharge.

C. Stationary or worse. Here the sedimentation rate started at 31 and ended at 32.

They point out the fallacy of taking an isolated sedimentation reading, as the rate is affected by many conditions, such as pleurisy, development of fluid in pneumothorax cases, worry, colds, teeth extraction, etc. They regard a patient with a sedimentation rate of over 30 per cent. as unlikely to improve under ordinary sanatorium treatment. Failure of

the sedimentation rate to improve under treatment suggests a bad prognosis, or that the treatment is not being successful, and new methods should be tried.

Professor Cummins (4) has published some investigations into the value of the sedimentation test and Arneth count. He writes : " While neither the sedimentation test nor the nuclear index provides new information unobtainable by ordinary clinical methods they afford two additional methods of measuring the constitutional balance of a case before, during and after treatment."

He carries out the sedimentation test with a small volume of blood obtained from a pricked finger. For the Arneth test the nuclear divisions of 100 successive polymorphonuclear leucocytes are counted and the total recorded.

Dr. Heaf (5) uses a larger quantity of blood, and he describes his technique as follows : " 0·4 c.cm. of a 3·8 per cent. solution of sodium citrate in sterile distilled water is drawn up into a 2 c.cm. syringe. The patient's vein is then punctured and 1·6 c.cm. of blood withdrawn. The contents of the syringe are placed slowly into a small gallipot, care being taken that the apparatus is dry and to avoid air bubbles in the citrated blood. A pipette holding 1·25 c.cm. graduated in 200 divisions is taken and the citrated blood thoroughly mixed. It is finally sucked up to the 200 mark on the pipette, care being taken to obtain a column of blood without air bubbles. The pipette is placed in a vertical position in a stand. Readings of the height of the column of red blood corpuscles are taken after the 1st, 2nd, 3rd, 4th, 6th, 9th, 12th, 18th and 24th hours." He claims that this method allows a series of readings over twenty-four hours to be taken, and that the bore of the pipette is so wide that if a number of red cells adhere together the clot will not affect the sedimentation rate, as is the case in a capillary tube.

With reference to prognosis Dr. Heaf found that the rate of sedimentation was increased in cases in which the disease was obviously progressing, and decreased in cases in which

the lesion was becoming arrested. Of 150 patients whom he watched for from six to ten months it was found that prognosis based on a series of sedimentation tests was correct in 82 per cent.

Vital Capacity.—The vital capacity is definitely lowered in pulmonary tuberculosis, so that a normal finding may be taken as useful evidence against this disease, although a positive one is no evidence in its favour. The vital capacity increases if the patient improves and decreases if he becomes worse, and is therefore a good method of observing the effects of treatment. It is also of value in prognosis, especially if repeated readings are taken. It is very necessary to take great care in obtaining the vital capacity or the readings will be inaccurate. I use the spirometer of C. Verdin, made by Boulitte in Paris.

The patient should be seated comfortably in such a position that he cannot see the dial of the spirometer. He should inspire to his fullest capacity and then expire completely but slowly into the mouthpiece of the spirometer. At first many patients fail to take a full inspiration, or to empty their lungs completely, but with a little practice they understand what they are required to do. The first two or three readings should be ignored and the patient encouraged to try again, until it is found that each time he gives the same vital capacity. It has been stated that patients can increase their vital capacity with practice, but this is not really the case, and it will be found that the capacity for a given patient is remarkably constant when once he has been taught what to do.

Professor Dreyer showed that definite relationships exist between vital capacity and certain body measurements, and he published (8) a book containing tables showing what the normal capacity should be under various conditions. With Professor Dreyer (9) I studied vital capacity in relation to pulmonary tuberculosis and found that of 267 cases with tubercle bacilli in the sputum the vital capacity was above

normal in one only. This patient had tubercle bacilli discovered in the sputum once when he had a cough following a cold ; they were not found at any subsequent examination. X-ray and physical examinations failed to reveal any sign of disease, and he returned to work and kept well without developing any signs of clinical tuberculosis.

In our series it was found that vital capacity does diminish as the extent of the disease increases, but still more does it diminish with increasing toxæmia.

The following case shows the gradual decline in vital capacity as the patient grows worse :—

1920.	March.	Vital capacity .	— 33·1 per cent.
	May.	,, .	— 36·0 ,,
	September.	,, .	— 37·2 ,,
	December.	,, .	— 41·4 ,,
1921.	February.	,, .	— 52·0 ,,
	May.	,, .	— 52·0 ,,

The patient died in June, 1921.

The following two readings were taken from an acute case. The patient became ill in February and died in July. In March the vital capacity was — 16·9 per cent., in May it was — 62 per cent.

The following readings were taken from a chronic fibroid case, and it will be noticed how constant they remain whilst the condition of the patient kept practically the same :—

1919.	April.	Vital capacity .	— 24·5 per cent.
1921.	May.	,, .	— 21·0 ,,
1922.	March.	,, .	— 20·2 ,,

Even in advanced cases of fibrosis the vital capacity remains constant provided the condition of the patient remains unchanged, as the following readings indicate :—

1919.	April.	Vital capacity .	— 50·0 per cent.
1920.	February.	,, .	— 50·6 ,,
1921.	April.	,, .	— 50·7 ,,
1922.	March.	,, .	— 51·0 ,,

A good prognosis is suggested when the vital capacity is nearly normal, improves under treatment, or remains constant whilst the patient is leading an ordinary life.

A bad prognosis is probable if the vital capacity falls as soon as the patient begins to take exercise, and the outlook is still more serious if the capacity gradually becomes worse during treatment. A low vital capacity is produced by many other conditions besides tuberculosis, and provided it remains constant it does not necessarily indicate a bad prognosis.

Complications.—The development of certain complications, such as meningitis or enteritis, make the prognosis hopeless. Toxic diarrhœa must be distinguished from ulceration of the intestine. Tuberculous peritonitis occurring during tuberculosis of the lungs usually proves fatal if accompanied by continuous pyrexia. Patients often live for many months, but it is a far more serious condition than tuberculous peritonitis occurring in a patient whose lungs are healthy. Tuberculous laryngitis was formerly thought to be a fatal complication, but although it undoubtedly adds to the gravity of the disease under modern treatment many patients recover.

Hæmoptysis rarely proves fatal, and it has been said that patients do not die of hæmoptysis, but after it. The after-effects, and especially tuberculous broncho-pneumonia, are certainly more dangerous than the hæmorrhage itself. Some patients are liable to repeated attacks of hæmoptysis, and many of them live for years, and, except that they have to rest for a few days during each attack, they maintain a good standard of health. The prognosis is always uncertain in such cases, owing to the possibility of an acute exacerbation following hæmoptysis. A sudden or large hæmoptysis is not common, but more often occurs in the chronic fibrotic type of case with large cavities than in other types of the disease.

Hæmoptysis may actually improve the prognosis by

drawing attention to disease which might not otherwise have been detected, and more especially by alarming the patient, and thus making him willing to undergo thorough treatment at the beginning of his illness instead of waiting until the disease has passed into the incurable stage.

REFERENCES

(1) Milne, Colin. *B.M.J.*, 1928, *ii.*, 1173.
(2) Fowler, Sir J. Kingston. "Pulmonary Tuberculosis," p. 186. London : Macmillan & Co., 1921.
(3) Trail, R. R., and Stone, D. M. *Lancet*, 1929, *i.*, 179.
(4) Cummins, S., Lyle and Acland, C. M. *Tubercle*, 1927, **IX.**, 1.
(5) Heaf, F. R. G. *Tubercle*, 1926, **VIII.**, 97.
(6) Drolet, G. T. *Am. Rev. Tub.*, 1924, **X.**, 280.
(7) Ford, A. P. *Tubercle*, 1925, **VII.**, 117.
(8) Dreyer, G., and Hanson, G. E. "The Assessment of Physical Fitness." London : Cassell & Co., 1920.
(9) Dreyer, G., and Burrell, L. S. T. *Lancet*, 1920, *i.*, 1912 ; *ibid.*, 1922, *ii.*, 374.

CHAPTER VI

CERTAIN COMPLICATIONS

Hæmoptysis

Much has been written about the treatment of hæmoptysis and the number of remedies advocated bears testimony to their failure.

The first point to remember when confronted with a case is that it is a most alarming occurrence for the patient and his friends, and it is necessary to reassure them and give them confidence.

The second point is that, although something definite must be done to calm the patient, some methods of treatment are actually harmful, and even if one cannot stop the hæmorrhage one can at least avoid making it worse.

The bleeding may vary from a mere staining of the sputum to a large and fatal hæmoptysis, although such a serious one is rare. Patients do not die of hæmoptysis, they die after it as a result of its complications. A large hæmoptysis usually comes from a ruptured vessel or an aneurysm of the pulmonary artery in a cavity, a small one from capillary congestion. The blood may come from the systemic (bronchial vessels) and not from the pulmonary circulation, and in any case pulmonary pressure is not of the first importance, since whether raised or lowered the same quantity of blood has to pass through the pulmonary system, as all blood leaving the left ventricle has to go through the lungs and right side of the heart.

Drugs given for the purpose of affecting the pressure are of doubtful value therefore, and they would probably be actually harmful but for the fact that if they do have any

effect on the blood pressure at all it is very transient. Drugs such as amyl nitrite, adrenalin, pituitrin and ergot are best avoided. Adrenalin is said to constrict the peripheral arteries except the pulmonary, which it does not affect, and the cerebral and coronary arteries, which it causes to dilate ; pituitrin is said to lower the pulmonary but raise the general blood pressure. In practice, however, no effect is produced on hæmoptysis. Turpentine is harmless in small doses but poisonous in large ones, and even large doses taken by the mouth have no effect whatever on hæmoptysis. Cold drinks or sucking ice have been advocated, but they dilate the pulmonary vessels and tend to increase the hæmorrhage. Hot drinks constrict the vessels, but have a stimulant effect and should be avoided. An ice-bag applied to the chest is said to constrict the vessels beneath it. I have never seen it have any effect on the hæmorrhage, but it may soothe the patient and so have a good mental effect.

Sodium chloride given by mouth (℥i. in water ℥v.) or intravenously (5 c.cm. of a 10 per cent. solution) has also been advocated and is said to increase the coagulability of the blood by drawing into the blood stream tissue fluid and so thrombokinase. If given by the mouth it may cause vomiting, which I think should be avoided, although some advocate emetics partly because they tend to clear the bronchial tubes and partly because the effort of vomiting may rupture the bleeding vessel and so, by allowing the ends to contract, stop the hæmorrhage.

Horse serum is quite useless in my experience, and I have not seen any good results from hæmostatic serum or coagulenciba, given subcutaneously. In one case I injected coagulenciba through the circo-thyroid membrane into the trachea, but it had no effect.

Calcium chloride increases the coagulation rate of the blood considerably. H. Elving (2) found the rate increased from five and half minutes to half minute in one case after the intravenous injection of 20 c.cm. of a 15 per cent. solution of

calcium chloride. Many cases have been described where hæmoptysis has stopped rapidly after such an injection, but it tends to stop automatically, and I have never convinced myself that this result was due to the calcium in any of my cases. Osler pointed out that treatment which might do good in a case of congested mucosa would be as much out of place in hæmorrhage from a ruptured pulmonary aneurysm as in a cut radial artery. However, any effect the calcium did have would be for the good and it is quite rational to use it in certain cases.

Morphia is advocated by one school and denounced by another in the treatment of hæmoptysis. Riviere (3) discusses the advantages and disadvantages of opiates and gives a very full account of the various methods of treating hæmoptysis. He says that the modern tendency is :—

1. To delay the use of narcotics in large hæmorrhages so that cough may be efficient in clearing the tubes.
2. To avoid their routine use.
3. To replace morphine by codein, heroin or dionin.

At the conclusion of his paper he points out that, after all, the outcome of hæmoptysis in any individual case is " on the knees of the Gods," and commonsense methods to assist Nature are all that can really be done apart from collapsing the lung. To this I would repeat my warning of the possibility of doing harm by over-treatment. Dr. Riviere says : " Nature's method will here be plugging of the area around with blood clot—until, soon, her concern for clear air tubes leads her to defeat these efforts by the induction of cough. How clear a passage can be steered under such circumstances between the Scylla of infarcted lung with its threat of pneumonia or subsequent tuberculosis, and the Charybdis of continuing hæmorrhage is for the physician to ponder, for he holds the fatal rudder, opium and its derivatives, in his hands."

One view is that hæmoptysis is not often fatal, and in the rare cases when it did prove fatal morphia would not have

saved the patient. On the other hand, tuberculous broncho-pneumonia or bronchitic spread of the infection does develop not infrequently after a hæmoptysis, and this is due to blood getting into the bronchi and remaining there. Treatment should be to clear the bronchi, whereas morphia tends to prevent cough. All this is perfectly true, but a small amount of morphia does not prevent cough and certainly does not lead to accumulation of blood in the tubes any more than natural sleep does, and it does undoubtedly pacify the patient. Most physicians will agree that it is bad practice to keep a patient deeply under the influence of morphia or to give an injection of morphia every time a fresh hæmoptysis occurs, but an initial injection at the onset does no harm whatever. On the contrary, it does a great deal of good by calming the patient and preventing restlessness. It must be admitted that blood should not be allowed to remain in the bronchial tubes, but too much coughing undoubtedly breaks clots which have formed over the bleeding surface and the violent expiratory and inspiratory efforts of cough prevent clotting and increase the hæmorrhage. In order to clear the tubes of blood some advocate the ambulatory treatment of hæmoptysis. The patient is allowed out of bed, given stimulants and even emetics. Whilst admitting the danger of broncho-pneumonia or spread of disease after hæmoptysis, I think it is far more dangerous to allow a patient to walk about and to encourage too much coughing.

In order to decrease the quantity of blood passing through the lungs the thighs have been bandaged sufficiently tightly to stop the venous circulation. After an hour and a half or two hours one arm is bandaged and one thigh unbandaged, and then the other arm is bandaged and the thigh unbandaged. Half an hour later first one arm and then the other are unbandaged. This method is also said to increase the thrombokinase in the blood, since the bandaging produces œdema, and on unbinding the limbs the tissue fluid

flows into the blood stream. A similar result is said to follow dry cupping the back or abdomen.

In a severe case of hæmoptysis artificial pneumothorax should be induced if one can determine from which lung the blood is coming: 300 or 400 c.cm. of air introduced into the pleural cavity are usually sufficient to stop the bleeding. Formerly I used to give a large initial quantity of air, but now I do not think it necessary. Should the first injection fail to check the hæmorrhage it is easy to give another one, and if too much air is given at once a reaction is apt to occur and add to the patient's discomfort, even if no harm is done.

One patient, a girl of eighteen, had slight hæmoptysis in August, 1920. She quickly recovered, but was found to have a lesion at the apex of the right lung and T.B. were found in the sputum. On December 18th hæmoptysis returned and she coughed up 5 oz. of blood, on the 19th 8 oz., and on the morning of the 20th another 8 oz. In the afternoon of the 20th she was coughing up a quantity of blood and a right artificial pneumothorax was induced: 1,400 c.cm. of air were given and the intrapleural pressure was raised from −11 −8 to −1 + 5. She had no further hæmoptysis. The first refill was given two days later, the first few injections being as follows:—

December 20th, 1920.		−11	−8	1,400	−1 +5
,,	22nd ,,	−11	−8	1,200	0 +3
,,	26th ,,	− 3	−1	900	+4 +6
,,	30th ,,	− 4	−1	700	+5 +7

After the initial treatment she had a reaction with a temperature of 103, but it fell to normal in thirty-six hours. A slight reaction followed the first and second refill, but after that there were no reactions and the temperature remained normal. She is now (January, 1929) alive and at work, but has signs of fibrosis in the upper zone of the right lung.

In cases of repeated hæmoptysis artificial pneumothorax

may be successful. One of my patients, a man of twenty-three, had hæmoptysis in 1906. He had recurrent attacks at intervals of about a year until 1918 when they became more frequent. In 1921 he was having hæmoptysis up to half a pint every few weeks. In one month he had three attacks and he had been unable to work for over a year. In March, 1921, during an actual attack of hæmoptysis artificial pneumothorax was induced on the right side. This stopped the bleeding and he is now (January, 1929) well and has had no more hæmoptysis. The lung was kept collapsed until July, 1922, but he returned to work in May, 1921, and attended every three or four weeks for refills.

In some chronic cases it is impossible to induce a pneumothorax owing to adherent pleura, and under these circumstances thoracoplasty should be considered.

The treatment of hæmoptysis must, of course, depend on its severity, but the following may be suggested as a basis for treatment.

Put the patient to bed and keep him propped up with pillows in the semi-upright position. A day and night nurse should be employed and no visitors should be allowed, although it may be necessary to let certain members of the family see him. The patient must be reassured, for however well he may control his feelings he is certain to be very alarmed and anxiety leads to restlessness and a quickened heart-beat. In some cases the physician is able to pacify the patient by his personality and assurance that all will be well, but it is often best to give a small injection of morphia. This will tide the patient over the initial period of terror, will relieve restlessness and assist clotting to take place. It is not necessary to give enough morphia to stop cough and it is very rarely necessary to repeat the injection. I agree that morphia should not be given as a routine, but, in my opinion, it has a very real value in the treatment of certain cases of hæmoptysis. The nurse should be instructed to keep the patient as quiet as possible. Too much fussing should be

avoided. Cough should be encouraged if there is anything to be expectorated and it should be explained to the patient that there is a certain amount of blood in the tubes which has to be coughed up and that there is no reason to feel alarmed about it. On the other hand, the continuous dry, useless cough, which so many patients develop, should be checked—it is largely a matter of habit and the patient can usually control it. A good cheerful nurse who has the patient's confidence is of the utmost value.

The bowels should be kept open and an initial dose of 2 gr. of calomel, followed five hours later by a dose of salts, may be given. The diet should be light and not stimulating, but it need not be cold. Ice to suck or iced drinks are harmful. Fluid should be restricted and should not exceed a pint and a half daily during the actual hæmorrhage ; 8 oz. of pounded fish or chicken, or 5 oz. of rabbit, raw meat sandwiches, bread and butter, milk and eggs are suitable foods, and it is not necessary or even wise to keep a patient strictly on a milk diet.

No medicinal treatment is required in mild cases, but as a placebo to comfort a nervous patient it may be wise to give a mixture such as :—

Pot. Brom.	gr. xv.
Tinct. Hyoscyami. . . .	℥xv.
Syr. Aurantii.	℥i.
Aq. ad.	℥ss.

three times a day.

An icebag on the chest is harmless and may be ordered if necessary for its effect on the mental state of the patient.

By these methods the great majority of mild cases will soon improve. The patient should be kept in bed for at least three days after the sputum is free from all traces of blood, and in the event of bronchitis or fever following the hæmoptysis he should be kept propped up in bed and given an expectorant mixture. After-treatment must depend on

the condition of the patient and whether any activity of the tuberculosis has been set up by the hæmoptysis.

In cases of moderate severity, where the hæmoptysis continues for six or more hours, an intravenous injection of 10 c.cm. of a 10-per cent. solution of calcium chloride may be given. It is, of course, quite useless in a large hæmorrhage due to a ruptured pulmonary aneurysm, but may help in a case of congestion or oozing.

A better method of treatment for moderate or continued cases is to bind the limbs as already described.

In more severe cases artificial pneumothorax is indicated, and in any case where the bleeding continues for more than three days and there is no contra-indication a pneumothorax should be induced not only to stop the hæmoptysis but to lessen the chances of a subsequent tuberculous broncho-pneumonia.

Pleurisy

A. Dry Pleurisy.—Pleurisy is a very common occurrence during the course of pulmonary tuberculosis. Sometimes it is painless and it may be unsuspected until revealed by post-mortem examination. There may, however, be considerable pain in a case of acute dry pleurisy, and to relieve this the affected side should be rested as much as possible. In mild cases rest in bed with antiphlogistine or some other warm application over the painful part will be sufficient. Strapping the side with straps of adhesive plaster passing from the vertebral column completely round the chest to beyond the sternum, and applied when the chest is in a state of deep expiration, will rest the lung and alleviate the pain. It is essential to make certain that the strapping is put on evenly as if it becomes crumpled it is very uncomfortable. Sodium salicylate is frequently given, and a draught consisting of Phenazone, gr. x., Sp. Amm. Arom., ℥ss., Aq. Chlorf. ad ℥i., will usually relieve the pain. In severe cases, however, an injection of morphia is required to give relief. Cupping over

the painful area is often very comforting to the patient, and formerly leeches were applied if the pain was acute. In really bad cases, however, the induction of a partial pneumothorax is the best treatment and should always be considered in any case bad enough to justify giving morphia. It is not necessary to give more than 200 or 300 c.cm. of oxygen, which are quite enough to separate the visceral and parietal layers of pleura. When absorption of the oxygen occurs and the two pleural surfaces again come into contact the pain does not, as a rule, return, possibly because a thin layer of lymph has formed over the inflamed surfaces. Oxygen is preferable to air in these cases as it is more quickly absorbed, and if the pleural surfaces are kept apart for long an effusion is very likely to result. In cases where the underlying lung is affected and it is desired to maintain an artificial pneumothorax it is better to use air. In certain cases, of course, it is not possible to produce any collapse owing to adhesions, but in the majority of cases where a pleural rub can be heard a sufficient collapse can be obtained to relieve the pain and the relief is often dramatic. Even in pleurisy from malignant disease the pain may be stopped by pneumothorax. One of my patients with pulmonary neoplasm developed pleurisy which caused great pain, prevented her from sleeping and would not yield even to morphia. A very partial pneumothorax was induced and the pain immediately ceased and did not return throughout her illness although the lung quickly re-expanded and a coarse pleural rub could be both heard and felt.

In cases of continuous dragging pain, especially that resulting from diaphragmatic pleurisy or adhesions, phrenic evulsion may be successful, and its possibilities should always be borne in mind.

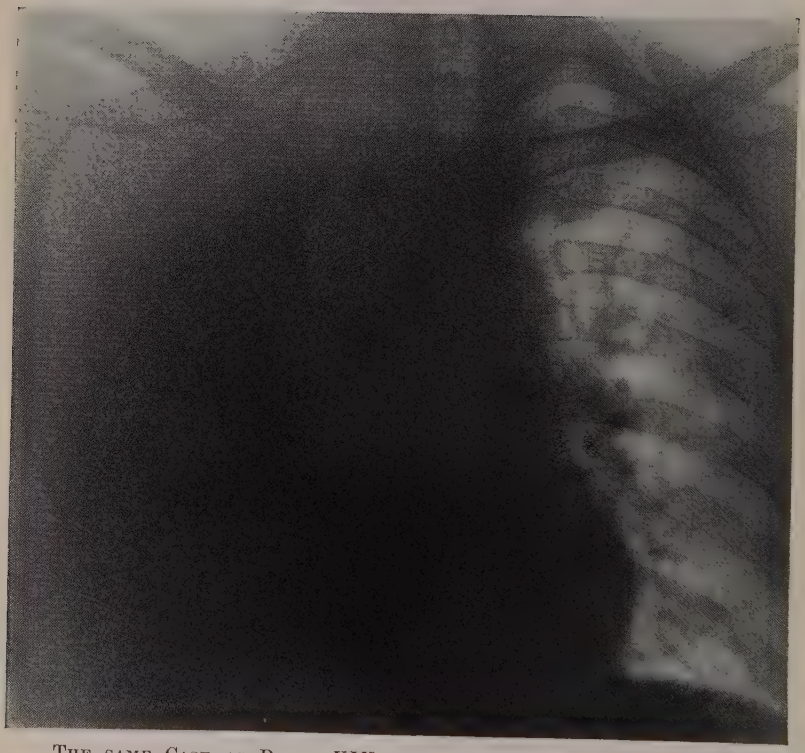
B. Pleural Effusion.—Clear serous fluid is usually straw-coloured, but may be tinged with red from admixture with blood. It may form in the pleural cavity as a transudate, the result of circulatory stasis, or as an exudate which

PLATE XIX

Right Pleural Effusion with the Patient in the Erect Position.

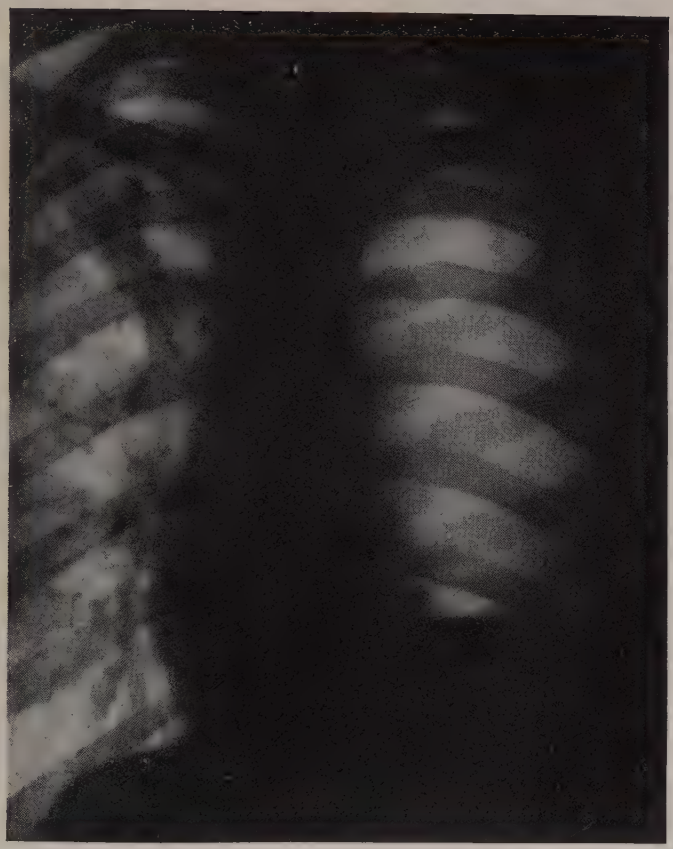
[*To face p.* 68

PLATE XX



The same Case as Plate XIX with the Patient recumbent.

PLATE XXI



HYDROPNEUMOTHORAX WITH THE PATIENT IN THE ERECT POSITION. NOTE THE FLUID LEVEL AND COMPLETE COLLAPSE OF THE LUNG.

[*To face p.* 68.

PLATE XXII

Same Case as Plate XXI in the Recumbent Position.

develops from inflammation. Lord (4), in a description of pleural fluid, says that a transudate and exudate differ, as shown in the following table :—

	Transudate.	Exudate.
Specific Gravity.	1010—1015 Hydræmic fluids having lower specific gravity than transudates from venous stasis.	1018 or more.
Albumin .	1—3 per cent.	4 per cent. or more.
Fibrinogen .	Trace.	Larger amount.
With acetic acid.	Very slight precipitate.	Bigger precipitate.
Coagulates.	Slowly or not at all.	Quickly.

It has been stated that when the predominant cells are small lymphocytes the effusion is due to tuberculosis, if polymorphonuclear leucocytes predominate the cause is some pyogenic infection (such as Pneumococcus, Streptococcus, Staphylococcus), whereas an excess of endothelial cells suggests an effusion of mechanical origin. Lord points out that the intensity of the pleurisy as well as the cause affects the cells in the effusion. Thus in long standing transudates the endothelial cells may diminish and the lymphocytes increase. A transient excess of polymorphonuclear leucocytes may occur in the early acute stage of a tuberculous effusion and a secondary infection in a chronic effusion will affect the cells. In malignant disease the cells of the effusion are similar to those found in transudates, endothelial cells being in excess. An excess of eosinophil cells in the effusion is regarded by some as evidence against tuberculosis.

Tubercle bacilli are not found in the early stages, but in

the long-standing tuberculous effusions they are usually to be found and often in large numbers. Inoculation of a guinea-pig will often reveal the presence of tuberculosis, although the bacilli could not be found microscopically, but even this test is usually negative in the early stages of the effusion.

Treatment.—As soon as an effusion develops it is safest to remove some 5 c.cm. for examination in order to confirm the diagnosis and exclude empyema. If the fluid is clear and is of tuberculous origin it is best to allow a natural absorption, which usually occurs in the course of a week or two. No medicinal treatment is required except to relieve the symptoms, such as constipation, sleeplessness, etc., but the patient should be kept in bed until the temperature is normal and the fluid sufficiently absorbed for the heart and mediastinum to be in the correct position. Some patients with clear tuberculous effusions run a high hectic temperature for several weeks, but it eventually settles and does not indicate the onset of empyema. An intermittent or hectic temperature, which alters and becomes a high continuous one, is of serious omen, as it usually indicates the development of miliary tuberculosis.

Aspiration should be performed in these tuberculous cases only if the effusion is so large that it causes considerable dyspnœa or pressure symptoms or if it is desired to convert the effusion into a pneumothorax (5). If there is no sign of absorption after three weeks it is usually best to remove the fluid and leave a pneumothorax.

In a large effusion causing pressure symptoms absorption may take place after some 8 or 10 oz. have been aspirated, but if it is decided to remove more than 15 oz. some air or oxygen should always be introduced to replace the fluid withdrawn. If this is not done the sudden expansion of the lung is apt to set up an acute tuberculosis by activating a quiescent lesion. Moreover the rapid lowering of the intra-pleural pressure may cause œdema of lung, faintness, or even sudden death. Oxygen should be used to replace the

fluid if one wants the lung to expand in a day or so, but air is better if it is desired to maintain a pneumothorax.

In order to replace the pleural fluid with gas the author's

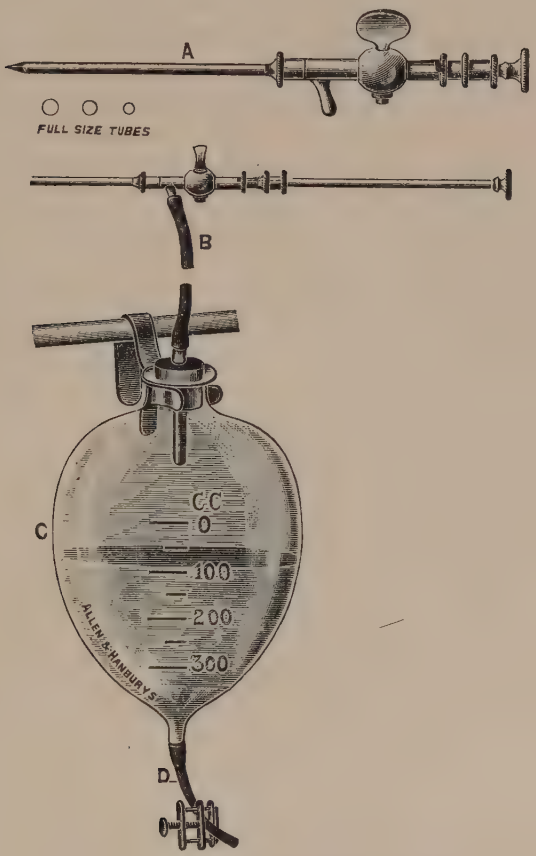


Fig. 10.—Aspirator used for gas replacements.

aspirator (Fig. 10) may be used. It has not the same suction power as Dieulofoy's piston or Potain's vacuum aspirator, and so for cases of simple aspiration I use Dieulo-

foy's apparatus. With Potain's the effusion is sucked into the exhausted bottle with a series of spurts and it is impossible to get an even flow of fluid with it. For gas replacement the author's aspirator has the advantage of working without attention when once started, and is therefore useful if the physician is single-handed, for he does not have to occupy himself with pumps, etc. Moreover the flow of the fluid is even and it is possible to keep the intrapleural pressure absolutely constant throughout a gas replacement. The aspirator consists of a trocar and cannula (A) made in three sizes and a graduated reservoir (C). The neck of the reservoir is fitted with a rubber cork through which a short glass tube is passed. This tube is connected with the trocar and cannula by a rubber tube (B). The contents of the reservoir can be allowed to flow out through another tube (D) into a suitable receptacle. To use the apparatus the tube (D) is closed, the reservoir partly filled with water and hung at the side of the bed. The rubber tube (B) is then connected with the side branch of the trocar and cannula which is then ready to be inserted into the pleural cavity. The patient should lie on his healthy side with the axilla of the affected side uppermost. After cleansing the skin over the back just below the angle of the scapula and anæsthetising the site to be punctured with 2 per cent. novocain solution the trocar and cannula is inserted into the pleural cavity. The trocar is then withdrawn, the tap turned off, the clip on tube (D) unfastened and the negative pressure produced by the flow of water through D will suck the pleural fluid into the reservoir (C). A needle connected with the pneumothorax apparatus is now put into the pleural cavity through the axilla and air or oxygen allowed to flow in. As this is the uppermost part of the body, with the patient lying on his side, the gas will collect and form an air space in the axilla as the fluid flows out through the lower needle.

I think it is better to use two needles as described, but the procedure can be carried out with one needle. In order to

do this when the trocar has been withdrawn and the tap turned off, the rubber tube from the pneumo-thorax apparatus is then fitted onto the cannula, and by clipping tube (B) and opening the tap air can be let into the pleural cavity, but one cannot insert air and remove fluid at the same time. Dr. Nelson (6) describes a method of gas replacement used in Pavia which appears very efficient but requires rather cumbersome apparatus.

After gas replacement the treatment is the same as an ordinary case of artificial pneumothorax complicated by effusion.

If after a simple aspiration the fluid re-accumulates sufficiently to necessitate a second aspiration, it is best to introduce a little oxygen into the pleural cavity. Repeated re-accumulation of the effusion should suggest the possibility of neoplasm, especially endothelioma of pleura.

Auto-serotherapy has been practised by some : that is, the subcutaneous injection of 1 c.cm. of the exudate directly it has been aspirated, but I do not think this method is of any value.

If, instead of clear fluid a tuberculous empyema forms, the treatment is different and the condition much more serious because :—

1. There is a tendency for great thickening of the pleura to occur so that the lung cannot expand and close the empyema cavity.

2. Serious complications, such as rupture of the empyema into the lung, are liable to occur.

3. Sinuses are apt to form through the tracks of the aspirating needles.

The treatment consists in emptying the empyema cavity by aspiration, and if the lung shows no sign of re-expanding in closing the cavity by thoracoplasty. The condition is especially frequent in cases of spontaneous pneumothorax, and treatment is discussed under that heading.

Spontaneous Pneumothorax

This may occur suddenly or gradually. In some cases it produces a sudden violent pain and severe dyspnœa, which may rapidly prove fatal. I saw one case mistaken at first for a perforated gastric ulcer. In other cases a patient who complains of dyspnœa for several days, but not enough to prevent him from working, is found to have a complete pneumothorax. Sometimes the condition is found accidentally by routine clinical or X-ray examination in a patient who has no symptom suggesting it.

In many cases the lung quickly re-expands and no treatment beyond a few days' rest is required. If the symptoms persist, however, and there is no sign of re-expansion after a week, it is best to remove some of the gas from the pleural cavity and so assist the lung to begin to expand. The gas should be removed gradually and no more than 700 c.cm. taken off at the first aspiration, otherwise a paroxysm of cough, severe dyspnœa and frothy expectoration are likely to occur.

In the severe acute cases where there is urgent dyspnœa gas should always be aspirated. It is often under great pressure and can be heard rushing out when the needle is put into the pleural cavity. The perforation in the visceral pleura is sometimes valvular, so that the air, which is forced into the pleural cavity when the patient coughs, cannot escape, and the intrapleural pressure consequently becomes very high and may lead to great displacement of the heart and mediastinum. In such a case the aspirating needle should be left in the pleural cavity with a long piece of rubber tubing leading from the needle to a bowl of water on the floor. This forms a water valve which allows the gas to escape but prevents any air from entering the pleural cavity.

In some cases there is considerable dyspnœa, but only a low intrapleural pressure, and in these there is usually a

nervous element so that the symptoms quickly subside after the patient has been given a small dose of morphia.

Tuberculosis accounts for over 80 per cent. of all cases, and now that X-ray examinations are so frequently made it is known that a partial pneumothorax is quite a common occurrence in cases of chronic pulmonary tuberculosis. In the great majority of tuberculous cases fluid forms and is apt to develop into tuberculous pus. In non-tuberculous cases, however, an effusion rarely forms, and as a rule the lung quickly re-expands without leaving any bad results. This agrees with the findings of artificial pneumothorax, where over half of the tuberculous cases develop some fluid during treatment, but the non-tuberculous cases practically never get an effusion.

When a spontaneous pneumothorax is followed by the development of tuberculous pus in the pleural cavity the patient almost always dies of the disease sooner or later. Tubercle bacilli are often absent at first in the effusion, but later appear in large numbers and a secondary infection is liable to occur. Repeated aspiration is apt to cause sinuses and make the prognosis still more grave. Even if the original perforation in the visceral pleura does heal the pleura tends to become very thick and to prevent re-expansion of the lung so that the pleural cavity is nothing more than a bag of tuberculous pus. Every effort should be made therefore to re-expand the lung. As soon as it becomes clear in such cases that the lung cannot expand I advise thoracoplasty, for the longer the operation is delayed the worse chance for the patient. This applies to all cases of tuberculous empyema, and not only those which follow spontaneous pneumothorax.

In order to stimulate re-expansion of the lung the pus should be aspirated completely and the intrapleural pressure left negative. If the pus is thick 15 or 20 c.cm. of Gauvain's modifying fluid (guaiacol, 2 grm., creosote, 2 grm., iodoform, 5 grm., ether, 10 grm., sterile olive oil, 100 c.cm.) may be injected into the pleural cavity a day before aspirating to

render the pus more liquid. Of course if the perforation in the visceral pleura is open the patient will taste this modifying fluid, and failure to taste it may be taken as evidence that the perforation has healed and therefore that there is more likelihood of the lung re-expanding.

The pleural cavity may be washed out with normal or hypotonic saline or with a 1 in 4,000 solution of methylene blue. In order to do this two needles are used as in gas replacement and the saline is introduced through a funnel

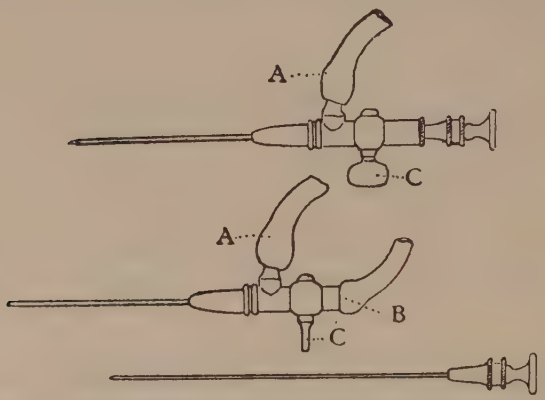


Fig. 11.—Needle with tubing as used for fluid replacement.

and rubber tube attached to B (Fig. 11) of the pneumothorax needle which is inserted into the chest through the axilla. When the tube (A) leading to the pneumothorax apparatus is clipped and tap (C) turned on the saline will flow into the chest and pass out through the aspirating needle in the back. The position of the patient should be changed from time to time in order to mix the pus with the saline and the pleural cavity washed out until the fluid leaving through the lower needle is quite clear. Tap (C) should then be turned off, tube (A) unclipped so that the intrapleural pressure can be ascertained and adjusted by introducing air or oxygen whilst the remainder of the fluid is being aspirated through

the lower needle. If there are sinuses or if there is much toxæmia it is best to wash out the pleural cavity in this way once or twice in order to get it as clean as possible before thoracoplasty and to improve the patient's general condition.

The following case illustrates the treatment in a case where the original perforation did not heal and thoracoplasty was performed.

The patient, a man of thirty-three, developed a right spontaneous pneumothorax in September, 1925. On November 3rd, 1,750 c.cm. clear fluid were removed from the right pleural cavity. It was sterile and no tubercle bacilli were found in it. On November 19th, 200 c.cm. of turbid fluid were removed. In January, 1926, 1,000 c.cm. of turbid fluid were removed, and in March, 1,200 c.cm. of greenish pus were aspirated. In April another 1,200 c.cm. of pus were aspirated and it now contained tubercle bacilli in large numbers. There was still an open perforation in the visceral pleura, as was shown by the patient tasting the modifying fluid which was introduced into the pleural cavity, and as there was no sign either by clinical or X-ray examination of the lung re-expanding thoracoplasty was advised. This was done in two stages and a month later phrenic evulsion was performed. The result was very satisfactory, the pleural cavity being obliterated and the patient making a good recovery.

The operation is not free from danger, especially in cases where there are sinuses, but I believe the patient is in far greater danger if left alone. If the perforation in the visceral pleura remains patent the danger is considerable, not only of the pleural cavity being infected but of dissemination of disease in the lungs. In one case of spontaneous pneumothorax, when some lipiodol was introduced into the pleural cavity, X-ray showed traces of it in both lungs on the following day, suggesting that the lipiodol, and consequently the tuberculous pus also, is aspirated into both lungs by the inspiratory efforts which accompany cough. Under modern

conditions, and in the hands of a surgeon accustomed to this type of work, the operation has not the same terrors it possessed a few years ago, and I feel convinced that it is more often advised too late than too early in these cases. The reason for this is that so many cases do well for a time and appear to be recovering when an accident, such as rupture into the lung or infection of the cavity as shown by the beginning of pyrexia, return of cough or sputum, leads to a steady or rapid decline in health and ultimately renders thoracoplasty essential. West (7) gives the general mortality from spontaneous pneumothorax as 70 per cent. In my opinion the following conclusions may be drawn :—

1. Spontaneous pneumothorax of simple or non-tuberculous origin usually heals quickly and is not complicated by the formation of effusion. If the lung does not re-expand after a few days some gas can be removed from the pleural cavity and this usually leads to re-expansion of the lung.

2. Spontaneous pneumothorax, followed by the development of effusion, is almost always of tuberculous origin. In some cases the fluid is absorbed and the lung re-expands with or without aspirating. If, however, the fluid turns into tuberculous pus the prognosis becomes much more serious and steps should be taken to cause re-expansion of the lung. If there is no sign of re-expansion thoracoplasty should be performed whilst the condition of the patient remains good. The operation is also indicated if the perforation in the visceral pleura shows no sign of healing after three months.

3. In some cases of pulmonary tuberculosis spontaneous pneumothorax is beneficial and it is then best to keep up an artificial pneumothorax by giving refills and so keeping the lung collapsed.

Bronchitis and Mixed Infections

When a patient with chronic bronchitis develops pulmonary tuberculosis the prognosis is very grave. In any case a liability to bronchitis in a consumptive is a serious com-

plication and necessitates special treatment. Such patients must be kept warm and it is harmful to try to harden them by getting them acclimatised to cold fresh air. Climatic treatment is indicated and the winters should be spent in a warm, sunny district, where the temperature is as constant as possible. If the patient cannot afford to go away for the winter he should be kept in well-ventilated but warm rooms and allowed out only when the weather is suitable.

It has been suggested that the tubercle bacillus itself is not very harmful, and that the serious symptoms in pulmonary tuberculosis are due to superimposed infection by pyogenic organisms. Attempts have been made to treat this mixed infection by anti-streptococcal, anti-pneumococcal and other sera and vaccines, but with no striking success.

REFERENCES.

(1) Burrell, L. S. T. *Lancet*, 1920, *ii.*, p. 242.
(2) Elving, H. *Tubercle*, 1922, **III.**, p. 323.
(3) Riviere, Clive. *Tubercle*, 1928, **IX.**, p. 509.
(4) Lord, F. T. "Diseases of the Bronchi, Lung and Pleura," p. 654. Philadelphia and New York, 1925.
(5) Burrell, L. S. T. *Lancet*, 1928, *i.*, p. 304.
(6) Nelson, T. S. 1924, **V.**, p. 265.
(7) West, S. "Diseases of the Organs of Respiration," p. 767. London, 1902.

CHAPTER VII

TREATMENT

The principles of treatment may be summarised as follows :—

1. Attention to the general health of the patient and treatment of troublesome symptoms and complications are necessary at all stages.

2. During the acute periods of the disease the object of treatment is to check the spread of the disease and for this purpose rest is essential.

3. When the acute stage is passed the object of treatment is to restore the tone of the body to its standard of fitness.

4. When the condition of the patient has been restored as far as possible the object of treatment is so to regulate his life that he may live and work within the limits of his impaired capacity.

I suggested (1) a classification based on the life history of the patient.

A. Stage of Institutional Treatment

1. **Hospital Stage.**—
   - (*a*) In bed, or up less than two hours a day.
   - (*b*) Up from two to six hours a day.

2. **Sanatorium Stage.**—
   - (*a*) Capable of less than half a day's work.
   - (*b*) Capable of half a day's work.
   - (*c*) Capable of a full day's work.

B. Stage of Maximum Improvement

(1) Capable of a full day's work.
(2*) Capable of part time work.
(3) Unable to work.

* This is the type best suited for life in some colony such as Papworth.

C. Period of Decline

(1) Able to do some work.
(2) Unable to work.
(3) Bedridden.

When the physician first diagnoses pulmonary tuberculosis he classifies the patient A1 *a*. Whether the treatment is actually given in hospital or in the patient's private house does not matter. Possibly the patient is found to have an acute tuberculosis and rapidly gets worse, in which case he would, of course, come into Class C3. On the other hand, it may be found that he can pass quickly through Classes A1 *a* and A1 *b* and become suitable for sanatorium treatment. The hospital stage is, therefore, that period of the disease during which the physician can observe the patient's response to treatment and can decide whether he is fit for sanatorium or not, and if so, for which sanatorium he is best suited. If he is not suitable he goes into Class B or Class C. I have purposely used the terms hospital stage and sanatorium stage because it is important to appreciate that the true functions of hospital and sanatorium are entirely different. The initial period of rest and observation is apt to be overlooked. There is many a sanatorium with a hospital side, and here patients can be treated during the acute or febrile phases of the disease, but treatment in a sanatorium is not the same as sanatorium treatment.

General Treatment

It is important to distinguish between treatment designed to improve the general health and that designed to check

the tuberculous lesion. For example, if an acute tuberculous joint is immobilised the splint is intended to deal with the actual disease, just as in pulmonary cases, to rest the lung by staying in bed, or more completely by pneumothorax, is to treat the disease. On the other hand, fresh air and regular living are intended to improve the general health and so indirectly affect the course of the disease. If a city worker is sent into the country, where he has good food and can lead a quiet regular life in the open air, his general health will improve just as will that of the consumptive under similar conditions. It is very important to appreciate this, because there is a popular belief that the treatment of pulmonary tuberculosis consists of living out-of-doors and overeating, and a patient is apt to think that no other treatment is required. It is not at all uncommon to find a patient, who has taken a few months' holiday and lived in a country cottage, playing golf and keeping as much in the fresh air as possible under the impression that he is doing the best for himself, whereas the disease steadily progresses. An initial period of rest will usually check the activity of the disease, and then a quiet country life may be beneficial.

Medicinal Treatment

This is of little value except to alleviate symptoms. Provided the patient is doing well he is better without any medicine at all except, perhaps, the occasional aperient which many people find necessary. Occasionally a tonic may help a patient and an acid strichnine mixture such as :—

Liq. Strych. Hyd. . . .	℥iii.
Glycerin	ʒss.
Ac. Nit. Hydroch. dil. . . .	℥xv.
Inf. Gent. Co. ad. . . .	ʒss.

taken before meals will be found useful. One must remember also that a certain class of patient has great belief in medicine, and a harmless mixture may produce a good

mental effect which is most important in such a chronic disease. Medicine should not, however, be prescribed as a routine.

Food

There can be no doubt that food is an important factor in treatment. Fat people are less liable to tuberculosis than are the thin, and in any condition where there is a shortage of food (for example, the Siege of Paris, 1870, and the Great War) the incidence of tuberculosis increases. It has also been stated that fat eaters are much less liable to become tuberculous than are those who dislike fat. It is undoubtedly a fact that, as a general rule, a gain in weight may be taken as a good sign and a loss is often the first indication of a breakdown. It is a mistake, however, to overfeed a patient, and provided he reaches his normal weight and maintains it no further gain in weight is required. The diet should consist of good plain food, and as far as possible one should avoid giving special articles of diet which the patient will not be able to obtain when he resumes his normal life. In ordering a diet it is necessary to take into consideration the type of food a patient has been in the habit of taking in the past and will have to go back to in the future.

Fat should be encouraged: mutton fat, bacon, butter, dripping, etc., may be advised, and most patients are able to take fat in some form or other. Milk is a good food, but there are substitutes, and it should not be forced on those with whom it disagrees. It is a mistake to get a patient into such a habit of taking extra milk that he is dependent on it.

Cod-liver oil is widely used and can be obtained in a tasteless form. It contains a fat-soluble vitamin of great potency, is very nutritious and especially useful in chronic cases who are under weight. It is quite unnecessary to use it as a routine, and it has been found at Midhurst Sanatorium that patients who are taking cod-liver oil regularly do no better than those who are not, although in special cases its value is admitted.

It is, of course, not only useless but actually harmful to give a patient more food than he can digest, and one often finds a patient who begins to improve and gain weight when the quantity of food is reduced, for he assimilates more and loses the chronic dyspepsia produced by over-eating. A weak digestion may be improved by fresh air, cheerful companionship during the meal, the inclusion in the diet of a little appetising food, even if not in itself nutritious, such as smoked salmon, a bitter tonic or, still better, a little alcohol. The question of alcohol and tuberculosis is a difficult one, for the disease is very chronic, and patients are so liable to periods of depression that the alcohol habit may well be formed. I do not think alcohol should be used in tuberculosis as readily as in certain acute diseases, where its action as a food alone, apart from its other effects, renders it a most valuable drug in helping through a crisis. Apart from the danger of developing a habit, alcohol in moderation is good. It undoubtedly assists digestion, and the addition of a glass of stout or port to the diet will often turn the scale in favour of the patient. Some years ago the routine treatment of pulmonary tuberculosis was rum and milk, and I am told that patients thrived on it. As a general rule, one would say that if patients are doing well without alcohol so much the better because of the real danger of producing a habit, and that spirits should be forbidden except in the case of elderly patients who are accustomed to them. On the other hand, one should never forget that alcohol often does good and should not be withheld under certain conditions.

During the acute febrile stage the diet should be as generous as is consistent with the patient's powers of digestion. Milk is often well tolerated but may lead to diarrhœa with offensive stools, in which case a more solid diet should be given and milk stopped.

It is in the subacute or sanatorium stage that diet is chiefly important, for the patient is trying to regain his lost strength and energy. Towards the end of sanatorium treat-

ment, however, extra or fancy diets should be dropped as far as possible so that the patient may not suffer when he returns home and has to live on an ordinary diet. Special diets have been advocated for tuberculosis but their value is doubtful, and although the patient should learn to eat wholesome food and avoid excess it is usually a mistake to encourage fads about foods. Some diets are designed to supply calcium and assist in calcification, or to make up for a calcium deficiency which has been noticed in tuberculosis. It has been suggested that a salt-free diet should be given as salts tend to produce an excess of fluid in the body. A diet without salt, however, is very distasteful to most patients and it has no practical value. I have not found any real advantage from the use of one of these special diets.

Rest

If one had to rely on one method only in the treatment of tuberculosis that method would have to be rest, for it is so vastly more important than all the other treatments. By resting the diseased part the lymph flow is impeded, there is a tendency to fibrosis and the disease becomes localised. These changes are seen in tuberculosis of any part of the body. The immediate improvement in a tuberculous joint when a splint has been applied, or in an acute pulmonary tuberculosis as soon as the diseased lung has been collapsed, is very striking. In any case of acute pulmonary tuberculosis an initial period of three or four months in bed is advisable. Before going to Frimley Sanatorium patients are admitted to Brompton Hospital, and I have found that those who have a long preliminary course of rest in the hospital do much better than those who go straight on to the Sanatorium.

As improvement takes place the patient requires a certain amount of exercise, and success of treatment is largely a matter of a correct balance between rest and exercise, and this is discussed in Chapter IX. dealing with sanatorium treatment.

Fresh Air

The value of fresh air is discussed in the same chapter. Although it is important at all stages of the disease, it should be regarded as part of the general treatment and not as a method peculiar to tuberculosis. Broadly speaking, one may regard the value of fresh air as two-fold :—

1. Those who work in the open air are, on the whole, more healthy than those who have indoor employment, provided they are physically fit to do their work.

2. Those who lead an open-air life (i.e., have windows always open and avoid stuffy atmospheres) are mostly immune to colds and catarrhal conditions. But a certain number of people are unable to stand cold and are harmed by a fresh-air life.

Treatment of Symptoms

Cough.—In many cases this is largely due to habit and patients can easily be trained not to cough. In the dining-room of any large sanatorium, where a number of consumptives are gathered together, a cough is seldom heard. Paroxysms of cough, which are due to difficulty in bringing up sputum and are especially liable to occur in the mornings, are often relieved by a simple mixture such as :—

Sod. Bicarb.	gr. xv.
Sod. Chlorid.	gr. v.
Sp. Chlorof.	℥x.
Aq. Anisi. ad	℥ss.

in an equal part of hot water. Patients with chronic disease who have tried many remedies often ask for this, as they get more relief from it than from any of the more complicated mixtures.

Other mixtures which help to loosen the sputum and relieve cough are :—

Ammon. Chlorid. . . .	gr. xx.
Ext. Glyc. Liq. . . .	℥i.
Glycerin	♏xx.
Aq. ad	℥ss.

or,

Pot. Iod.	gr. iii.
Pot. Bicarb.	gr. xv.
Aq. Camph. ad . . .	℥ss.

In the case of continuous cough which interrupts sleep a linctus should be given, such as :—

Oxymell. Scillæ . . .	℥ss.
Ac. Hydrocyan. dil. . . .	♏ii.
Morph. Acet.	gr. $\frac{1}{16}$.
Aq. ad	℥i.

or, if this fails, heroin will often give relief, and the following is a useful linctus :—

Heroin Hydroch. . . .	gr. $\frac{1}{16}$.
Glycerin	♏x.
Syr. Picis Liq. ad . . .	℥i.

It often happens that a patient gets accustomed to a linctus and it is then best to change it for a time. The following prescriptions may be found useful :—

Bromoform	♏$\frac{1}{2}$.
Terpin Hydrate . . .	gr. i.
Alcohol (90 per cent.) . .	q.s.
Heroin Hydroch. . . .	gr. $\frac{1}{40}$.
Tinct. Pruni Virg. . . .	♏iii.
Glycerin ad	℥i.

or,

Nepenthe	♏x.
Oxymell. Scillæ . . .	♏xx.
Syr. Pruni Virg. ad . . .	℥i.

Syrup Cocillanæ Co., which is prepared by Messrs. Parke Davis, will usually relieve an irritating cough.

For the useless, irritating cough, which does not produce sputum, a lozenge will often be sufficient. The Brompton Cough Lozenge (Trochisci Glycyrrhizæ, each containing Extract of Liquorice, gr. iii., and Anise Oil, ℥(½)) is efficacious, and the fact that patients who have tried many other lozenges frequently write for it shows that the patients themselves believe in its value. Cough is often caused by smoking, but under these conditions many patients prefer the cough.

Sputum.—Special treatment, such as artificial pneumothorax and sanocrysin, may be indicated if there is excessive sputum, and the quantity of sputum is often reduced if the patient gives up smoking. If there is sputum, however, it should be expectorated, and it is a mistake to give drugs such as opiates to suppress it.

Night Sweats.—These occur in the early acute stages or during an acute exacerbation in chronic disease. As the patient improves they almost always disappear, and it is uncommon to find them after a patient has had a few days' rest in bed. In some cases, however, they persist and may be so severe that the patient has to change his night clothes two or three times during the night. In such cases the patient should be given a tumbler of milk just before he goes to sleep, and if he wakes in the night he should take a little more milk, a few biscuits or some other food. This usually prevents night sweating, but in bad cases it may be necessary to have a special nurse to wake the patient periodically and give him a little warm milk. Omnopon, gr. ⅓, given the last thing at night, will sometimes allow the patient to sleep without sweating. A common remedy is a pill containing zinc oxide and belladonna, but I have found no benefit from it.

Pyrexia.—The best treatment for fever is rest. If rest in bed is not sufficient some further rest of the diseased part

by artificial pneumothorax, strapping the chest wall, or some other method may succeed. It is easy to reduce the temperature by drugs, but to do so is useless except for the mental effect on the patient who worries about his temperature, for it will rise again as soon as the drug is discontinued.

Dyspnœa.—This may be due to the extent of the pulmonary lesion or to the cardiac condition. In some cases when there is extensive fibrosis and the heart is much displaced relief may follow surgical treatment. In a few cases the removal of portions of two ribs thus leaving a flap will diminish the dyspnœa considerably. Relief in this case is probably due to the fact that during inspiration a large part of the lung is unable to expand and the force of the negative pressure affects the thoracic organs, including the heart which dilates. In some unilateral cases of fibrosis or obstruction to a large bronchus the heart does appear to dilate during inspiration. When therefore, portions of two ribs are removed, and a flap is left, the flap is sucked in during inspiration, thus saving the heart and relieving the dyspnœa. Phrenic evulsion may also be performed in such cases to relieve the dyspnœa.

Gastro-intestinal Symptoms.—These may be due to overfeeding and always necessitate a review of the patient's diet. Poor appetite and a weak digestion are very common and sometimes the initial symptoms of pulmonary tuberculosis. In a series of cases in which I had fractional test-meals done I found a deficiency of hydrochloric acid in the stomach in the majority, and I have obtained benefit by giving before meals a mixture containing :—

Lig. Strych. Hyd. . . .	ℿiii.
Glycerin	℥ss.
Ae. Nit. Hydroch. dil. . .	ℿxv.
Inf. Gent. Co. ad . . .	℥ss.

In most cases, however, the gastric symptoms disappear as the patient improves, and an abundance of fresh air is

the best stimulant to the appetite. Diarrhœa may be due to toxæmia without any ulceration of the intestine, or there may be actual ulceration, in which case there will be blood in the stools. In some cases, especially in the terminal phases of the disease, the stools become very offensive and loose, and this is usually due to the milk diet which is so commonly ordered in these cases. If the milk is altogether stopped and a solid diet substituted the stools will cease to be offensive, even in the worst cases, and the diarrhœa is usually considerably relieved. I have found no benefit from dimol, hyd. c̄. cret. or other intestinal disinfectants in these cases, nor have I obtained any results from the use of calcium chloride given intravenously.

CHAPTER VIII

TREATMENT—*Contd.*

Sanocrysin

Preparations of gold have been used for many years in the treatment of pulmonary tuberculosis, and sanocrysin is the form most commonly employed at present. Sanocrysin is a double thiosulphate of gold and sodium which was prepared by Moëllgaard, and was believed to destroy tubercle bacilli *in vivo*. It is given intravenously, and is frequently followed by a severe reaction resembling that seen after an injection of tuberculin. At first it was thought that sanocrysin killed a large number of tubercle bacilli, and that the reaction was due to the endotoxins which were consequently set free. This view was supported by the undoubted fact that tubercle bacilli did disappear or become very scanty in the sputum after injections of sanocrysin. An antitoxic serum was prepared and given before sanocrysin in order to counteract the reaction, but it proved of very doubtful value, and has now fallen into disuse. In 1890 Koch discovered that a preparation of gold cyanide had a bactericidal action on tubercle bacilli, but it was subsequently shown that, although a dilution of 1 in 1,000,000 prevented the growth of the bacilli in culture, it required a dilution of 1 in 25,000 to prevent their growth in serum. In the same way the action of sanocrysin on tubercle bacilli is affected by serum. Sweany and Wasick (1) tried the effect of sanocrysin on tubercle bacilli that had been kept in cultures for four years. There was blackening of the bacilli after about twelve days in all dilutions up to 1 in 20,000. There was complete inhibition of growth in dilutions up to 1 in

200,000, partial inhibition up to 1 in 500,000, and slight inhibition up to 1 in 1,000,000. But in a more recent strain of bacilli some colonies grew in dilutions of 1 in 20,000. After tubercle bacilli had been exposed for two hours to sanocrysin as strong as 1 in 1,000 in salt solution and serum, they grew on Petroff's medium quite well, and even after sixty days' exposure to 1 in 2,000 sanocrysin the culture injected into guinea-pigs produced tuberculosis. Wright (2) found sanocrysin 1 in 250 had no effect on tubercle bacilli in blood. Sanocrysin may produce shock by its poisonous action on the endothelium, but not by killing tubercle bacilli and producing a tuberculin reaction from the endotoxins.

At first when sanocrysin was used there were some disastrous results. About 16 per cent. died as a direct result of the treatment either from shock or acute metal poisoning. Antitoxic serum was used because it was then believed that shock was due to setting free endotoxins from killed bacilli. It was not successful, however, and its use has now been abandoned altogether by most physicians. As a matter of fact, the serum did not prevent genuine tuberculin shock. By decreasing the doses and lengthening the intervals between them these severe reactions were prevented. Professor Faber (3) advised dosage of 0·5 grm. intravenously for the first dose, and 1 grm. for subsequent doses, with intervals of three days between the first two doses and five days between the subsequent doses. If a reaction occurred he stopped the treatment until all signs of the reaction had gone. Smaller doses are now frequently used, but in my experience no effect is produced by very small doses. If a reaction occurs it is my practice to wait until all effects of it have passed, and then to repeat the same dose. I do not increase the dose if the previous injection has caused any reaction. If no reactions occur I gradually increase the dosage up to 1 grm., which I repeat three or four times. My initial dose is 0·1 grm.; after three days I give 0·25 grm., and then at weekly intervals 0·5, 0·75 and 1 grm. A course, therefore,

would last about six or seven weeks, and consist of 5·6 grm. or 6·6 grm. of sanocrysin.

In some acute cases I have given daily intravenous injections of 0·1 grm. sanocrysin for a week, and then after a week's interval repeated the course. In most cases this has had no effect for good or bad, and in the few where improvment has occurred I have not convinced myself that this was due to the sanocrysin rather than to the rest in bed.

Dr. Clarke (4) has written a full account of the uses and dangers of sanocrysin, and describes the result of treatment in 18 cases. In common with other observers, he noticed that the most striking effect was diminution in the quantity of sputum and the number of tubercle bacilli found in it. Sputum ceased altogether in 8 of his cases, was diminished in 4, and in the rest there was no change. He writes: "generally one or two tubercle bacilli could be found after prolonged search as long as any sputum was coughed up. On the other hand, there is no doubt that the number of bacilli tends to diminish very markedly. Two patients who ceased to have sputum for two months relapsed. When they began to produce sputum again tubercle bacilli in fair numbers were present."

In my series of sixty I divided the cases into five groups (5). All these patients had tubercle bacilli in the sputum before treatment.

1. Chronic fibro-caseous. This group contained 28 patients, and of these 19 improved, 8 remained unchanged, and 1 appeared worse as a result of the treatment. 15 lost tubercle bacilli from the sputum.

2. Cases where artificial pneumothorax had been induced for disease in one lung, and after an initial improvement relapse had occurred owing to spread of disease in the other lung. In this group were 10 patients; of these 9 improved and 1 became worse. 5 lost tubercle bacilli from the sputum.

3. Cases with active tuberculosis in both lungs. The worse

lung was collapsed by artificial pneumothorax, and sanocrysin was given for the other lung. In this group were 6 patients, of whom 5 improved and 1 remained unchanged. 4 lost tubercle bacilli from the sputum.

4. Acute pulmonary tuberculosis treated by sanocrysin without pneumothorax. There were 13 cases in this group. 5 of them improved, and 2 became worse. In 6 the course of the disease showed no change. Only 1 lost tubercle bacilli from the sputum.

5. A case of spontaneous pneumothorax and 2 of pleural effusion, with active tuberculosis in the non-compressed lungs. In 2 of these there was no change, and 1 became worse.

Dr. Clarke also noticed that in some of his cases there was an eosinophilia during the treatment, but it subsided after two or three months. In one of his cases there was an eosinophil count of 34 per cent. In many of his cases there was a fall in the Arneth count after an injection of sanocrysin, but in favourable cases the count rose after 24 to 48 hours, and remained high for some months. Those of his cases which did not show this fluctuation in the Arneth count did badly.

The sedimentation test is affected by sanocrysin. Dr. Trail (6) mentions eleven cases treated with sanocrysin because they were not doing well and still had tubercle bacilli in the sputum in spite of four months' treatment. The initial result of this treatment was a rise in sedimentation rate followed in satisfactory cases by a fall, which was as much as 20 per cent. in one case. Six of the eleven cases became T.B. negative. Dr. Heaf (7) found a similar initial improvement in the sedimentation rate, but in over 75 per cent. the rate increased again when the treatment was stopped.

The same fluctuation is seen in the vital capacity. After a sanocrysin reaction the capacity falls, but, if the patient is doing well under the treatment, it gradually rises and reaches a higher level than previously. The improvement after sanocrysin is often only temporary, and when a relapse occurs the

vital capacity will gradually fall as the disease spreads. A steady fall in the vital capacity is often one of the earliest indications of a relapse.

Indications.—It often happens that a patient improves under sanatorium or other treatment, and loses all symptoms except sputum containing tubercle bacilli. In these cases sanocrysin is especially useful, and will frequently cause the sputum to disappear after two or three injections, although relapses are common. In one case a man of fifty-five had chronic fibroid tuberculosis of seven years' duration, and there was X-ray evidence of cavities in both lungs. He was afebrile, and his general condition was good, but the sputum was loaded with tubercle bacilli. After the second injection of sanocrysin the sputum was much lessened in quantity, and tubercle bacilli were found with difficulty. After the fourth injection no bacilli were found, but otherwise his condition was quite unchanged. Two months later the sputum again contained numerous tubercle bacilli.

Patients with chronic pulmonary tuberculosis often have periods of comparative good health, alternating with periods when the disease progresses. Sanocrysin does not seem to prevent these relapses, but it does seem, in many cases, to check an exacerbation of disease and bring about a period of arrest. After an initial period of improvement many of my patients relapsed, but in some this was controlled by a second course of sanocrysin. An acute exacerbation in a chronic case is often checked by sanocrysin, but I have not found much result from the treatment in the primary acute cases.

In many patients with chronic fibro-caseous tuberculosis, the disease slowly progresses, and in these sanocrysin may be useful in checking the spread of the disease.

I have found sanocrysin very helpful in combination with pneumothorax in the treatment of bilateral disease. There are certain disadvantages in making a bilateral pneumothorax, and I generally prefer to collapse one lung fully and

give sanocrysin to control the disease in the other. I have now had eleven cases of this type, and eight of them improved and showed no spread of disease in the uncollapsed lungs.

The chief indications for treatment, therefore, may be grouped under four headings :—

1. To diminish the quantity of sputum and number of tubercle bacilli.
2. To check an acute spread of disease.
3. To treat a patient who is getting gradually worse under other treatment.
4. To treat bilateral disease in combination with artificial pneumothorax.

Complications.—In my series of sixty cases the following complications were observed during the treatment :

1. Febrile reactions. These can be divided into three groups :

   First, a sudden rise of temperature, starting an hour or less after the injection and lasting for a few hours. There is always a feeling of malaise during the reaction, and sometimes a rigor. This short reaction occurred in seventeen of my cases.

   Secondly, a rise of temperature which may start equally suddenly, but which may persist for a few days, is more severe than the first type of reaction, and is accompanied by malaise, headache, and sometimes vomiting. This type of reaction was seen in thirteen cases in my series.

   Thirdly, a long reaction which occurred in four of my patients. The temperature did not begin to rise until after the third injection of sanocrysin. The rise was gradual, and lasted for five days or more. In one case it lasted seventeen days, and was always accompanied by a slight amount of albuminuria, but with little or no malaise.

2. Albuminuria. A very faint trace of albumin was found

in most patients, but in only twenty-three was there any definite amount. There is a greater tendency for albuminuria to occur as the treatment continues, and this is one of the reasons why I do not give more than 6·6 grm. at any one course. After this quantity persistent albuminuria may begin. In most of my cases the condition was very mild and transient, and only in one case did I have to discontinue the treatment on this account. I should advise stopping after 5·6 grm. had been given in cases where more than the merest trace of albumin was found in the urine. In cases where the urea concentration test was made this was always found to be within normal limits in spite of the albuminuria.

3. Gastro-intestinal symptoms. Vomiting occurred directly after the injection in thirteen of my series, and was associated with a febrile reaction.

Looseness of the bowels was not uncommon, and a troublesome diarrhœa developed in two cases, but passed off in a day or two when the treatment was discontinued. Two patients had a mild stomatitis and eight complained of a metallic taste.

4. Aching in the limbs and rheumatic pains were present in seven cases. In one the joint pains lasted for several days, and were so severe that the sanocrysin injections were stopped.

5. An erythematous rash occurred in eleven of my series, and in three of these it was severe. In all cases where a rash appears I now stop the treatment. In one case a mild rash developed, and a week after it had subsided I gave another injection of 0·1 grm. sanocrysin, which was followed by a severe eczematous rash. The bad cases went on to a desquamative dermatitis, and, although in the end all did well as regards their pulmonary condition, the complication was by far the most serious in my series, and for this reason I should not advise continuing the treatment or starting another course in a patient who had developed a rash after a previous injection.

Results of Treatment.—In all my cases the patients were

having artificial pneumothorax, rest in bed or some other form of treatment, and it is impossible to give figures which would be of value in forming an opinion as to the exact results of sanocrysin treatment. If, after a course of sanocrysin, tubercle bacilli disappeared, and eventually the sputum cleared up altogether in two patients, but whereas one remained well the other relapsed after a few months, this would not necessarily mean that sanocrysin had succeeded in one case and failed in the other. In one case the defensive forces of the body were not able to take advantage of the check in the disease caused by the sanocrysin and make a permanent arrest, but the actual effect of the drug may have been exactly the same in both cases. Relapses are very common after this treatment, and in the majority of my patients the loss of sputum was only temporary. There can be no question, however, that sanocrysin does have a very distinct immediate effect on most cases. I have come to the following conclusions on the subject :—

1. Patients with chronic pulmonary tuberculosis who are failing to improve or becoming slightly worse under simple routine treatment do, in many cases, show a remarkable improvement as soon as they are given sanocrysin, and the most striking change is the diminution in the quantity of sputum and tubercle bacilli. In most cases of chronic pulmonary tuberculosis there are periods of quiescence and activity. Sanocrysin seems to cut short that period of activity and to precipitate one of quiescence or arrest, but it does not prevent the liability to relapse.

2. On an acute case sanocrysin often fails to produce any effect whatsoever, but sometimes will check the spread of the disease.

3. Sanocrysin should be regarded as a drug which, in certain cases, aids other methods of treatment. It is not a cure, and is never more than part of the treatment.

4. The dangers and complications of sanocrysin treatment are not great provided the dosage is carefully regulated.

Large doses are dangerous and small ones useless, so that it is necessary to adopt a medium standard of dosage.

5. Sanocrysin does not act by killing the tubercle bacilli in vivo. It probably has two actions, one which occurs only when sufficient has been given to produce a reaction or shock, and this is comparable to the action of other substances, such as T.A.B., or serum. The other, which is not connected with the ordinary result of shock, consists in stimulating certain parts of the body to destroy the bacilli. How this result is produced is not known, but sanocrysin does certainly differ from other substances so far employed in that when given intravenously to a consumptive patient the tubercle bacilli tend to disappear from the sputum in a very short space of time.

REFERENCES

(1) Sweaney, H. C., and Wasick, M. M. *Am. Rev. Tub.*, 1925, **XII.**, 316.
(2) Wright, Sir A. *B.M.J.*, 1926, *i.*, 102.
(3) Faber, Knud. *Trans. Nat. Assoc. Prevent. Tub.*, 1925, 157.
(4) Clarke, R. C. *Tubercle*, 1926, **VII.**, 473, 540 and 584.
(5) Burrell, L. S. T. *Tubercle*, 1928, **IX.**, 570.
(6) Trail, R. R., and Stone, D. M. *Lancet*, 1929, *i.*, 179.
(7) Heaf, F. R. G. *Tubercle*, 1926, **VIII.**, 97.

Serum.—Antituberculous serum has been used in the attempt to counteract tuberculous toxæmia, but has had no success. Various preparations of serum have been tried by different workers, but, although some have reported encouraging results, there is no real evidence that any particular serum has produced definite benefit, and the use of serum in tuberculosis has fallen into disuse.

As with tuberculin, if the dose of serum is increased to the extent that it produces shock, benefit may follow. I have, however, obtained the same result with normal horse serum.

Fig. 12 shows a case treated with antituberculous serum.

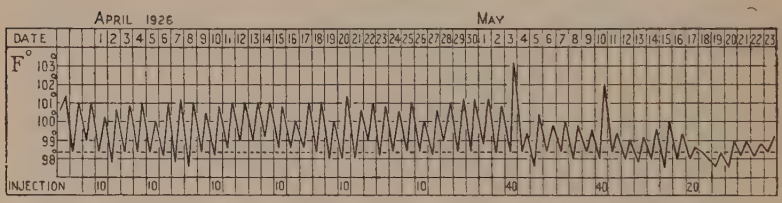


FIG. 12.—Temperature chart in a case of pulmonary tuberculosis treated by injections of serum. The figures at the bottom of the chart show the number of c.cm. of serum given on the various days.

The patient, a girl of twenty-one, had an acute exacerbation of pulmonary tuberculosis, and had six subcutaneous injections of the serum in 10-c.cm. doses without effect. When 40 c.cm. were injected she had a reaction, followed by a lowering of the temperature. After a second dose of 40 c.cm. a similar reaction occurred, and a week later it was decided to give her a third dose of 40 c.cm. On this occasion, however, when 20 c.cm. had been injected she had anaphylaxis, that is to say, she suddenly collapsed, stopped breathing, was pulseless, lost control of her bladder and rectum, became livid and appeared to be dead. She recovered, however, and from that time steadily improved. The temperature became normal, the pulse fell from 120 to 80,

tubercle bacilli disappeared from the sputum, and six months later she was in a sanatorium walking five miles a day.

In this case I believe the improvement was due to the shock, and not to any specific action of the serum.

Tuberculin.—Tuberculin has been used for many years in the treatment of tuberculosis. When it was first introduced there was a wave of enthusiasm, and it was regarded as a cure. The results were disappointing, however, and it appeared to make matters worse in many cases. Opinion began to swing to the other extreme, and tuberculin was looked upon not only as useless, but as actually dangerous. Since that time it has practically fallen into disuse, except by a few disciples, some of whom are lukewarm and others ardent in its praises. It is, however, a fact that after nearly fifty years the believers have failed to convince the world that tuberculin has any real value in treatment, and even in veterinary work it has not established its position as of therapeutic use. The arguments in favour of tuberculin may be summed up :—

1. Whatever may be the theories or results in animal experiments, tuberculin has been found to do good to cases of pulmonary tuberculosis in man.

2. Patients with incipient pulmonary tuberculosis treated with tuberculin fail to develop the disease, and a strong hypersensitiveness is corrected, so that after the treatment the patients fail to react even to large doses of tuberculin.

3. Some patients who have failed to improve under sanatorium or other treatment do improve with tuberculin.

In answer to these views it may be urged :—

1. The great majority of investigators have failed to observe any real benefit from tuberculin. Tuberculosis tends to cure itself, and a certain proportion of patients do get well without any treatment at all. Most observers have failed to convince themselves that this proportion is increased by the use of tuberculin.

2. Patients who are hypersensitive to tuberculin do not

necessarily develop clinical pulmonary tuberculosis. The so-called incipient type of case is just the type that does well under any treatment. In 1913 I collected twenty-three cases of children who were sent to my out-patient department at Brompton Hospital as suspected cases of tuberculosis and who had a positive Von Pirquet test. In 1920 all these children were traced and all were well, yet none of them had had any tuberculin treatment.

3. Some patients certainly do improve under tuberculin treatment, but others do not. It is possible to find a patient who has improved under any one treatment, though several other treatments have failed. Pulmonary tuberculosis is a disease that ebbs and flows. There are patients who failed to improve with tuberculin, but became rapidly better when it was stopped, and some other treatment started. When a patient with acute febrile tuberculosis goes to bed the temperature usually settles down quickly. If it does not do so the addition of tuberculin to the treatment does not cause it to fall. After a tuberculin reaction there is a tendency for the temperature to fall, but the same is seen after a reaction following non-specific protein such as T.A.B., or normal horse serum. In other words, the beneficial effect of certain treatment such as rest in bed, is plain, whereas the benefit of tuberculin is still a matter of debate.

I have seen many cases of tuberculin reaction, and in my experience the effects soon wear off, leaving no damage even after a severe reaction. Sometimes a serious and even fatal flare-up of the disease may be initiated by tuberculin, but the chances of this are remote, and it should not occur if the tuberculin is properly used. Any drug is dangerous in the hands of those who have no knowledge of its effects. I have seen good effects follow a reaction, and shall refer to this matter under Shock Therapy, but apart from shock or reaction I have failed to find a single case in which tuberculin could be clearly shown to have been of therapeutic value.

A few years ago Professor Dreyer prepared a defatted

tuberculin, which was known as diaplyte, and this was given a thorough trial under the auspices of the Medical Research Council, but the hopes that were entertained for it were not realised. Dr. Bardswell (1), after trying tuberculin at King Edward VII. Sanatorium, Midhurst, writes : " Collectively the results point to the conclusion that tuberculin when given in addition to the usual measures practised in the sanatorium had no appreciable effect either for good or ill."

I have formed the following conclusions regarding the therapeutic use of tuberculin :—

1. The improvement which occasionally follows tuberculin reaction may be seen after shock following the injection of other substances, such as normal horse serum. Apart from this action tuberculin has no therapeutic value whatever.

2. An overdose of tuberculin may do considerable harm, but it is a perfectly safe method of treatment if used by any one with experience. The dangers of tuberculin have been very much exaggerated, and any ill-effect is due to its misuse, and not its use.

REFERENCE

(1) Bardswell, N. *Med. Res. Council Special Report*, Series No. 33, p. 39.

Shock Therapy.—When tuberculin or live tubercle bacilli are injected into a sensitive animal there is an allergic reaction. A reaction may also occur when certain other substances are injected; for example, non-specific vaccine (such as T.A.B.), copper, gold, normal horse serum, etc. (1). These reactions are similar to anaphylaxis. In the case of tuberculin it requires but a small dose to produce a reaction in those who are sensitive to it, just as a specific protein will produce a reaction in a patient (with asthma, for example) sensitive to it. A tuberculin or allergic reaction is specific because it is not obtained in those who are non-sensitive, but the reaction does produce a shock very similar to that produced by copper or other substances, and very similar to anaphylaxis. I must emphasise that I do not deny that tuberculin has a specific action, and I do not suggest shock or reaction are the only effects of a given drug or vaccine. Sanocrysin, for example, tends far more than other drugs to cause diminution of sputum and disappearance of tubercle bacilli. My suggestion is that the shock by itself does have some effect on the disease. I have already described the case of a girl (p. 100) who had anaphylaxis whilst antituberculous serum was being given. The following cases will show under what various conditions a shock may occur and prove beneficial :—

Case 1.—A man of twenty-two developed signs of pulmonary tuberculosis early in 1921, and at the end of the year went into a sanatorium. He then had signs of disease all over the right lung, sputum loaded with tubercle bacilli, and an evening temperature of 102 F. Right artificial pneumothorax was started in December, 1921, but he remained ill and febrile until May, 1922, when he had a sudden reaction with increase of fever, and pleural effusion developed.

The sudden nature of this reaction and the fact that the intrapleural pressure went at once from — 6 to + 8 suggests that a perforation of the lung had taken place. Within a month not only did the effects of the reaction wear off, but

the temperature became normal, his whole condition gradually improved and he left the sanatorium free from symptoms in May, 1923. The pneumothorax was kept up until May, 1924, and since then he has kept perfectly well. Now, nearly five years later, he is still free from symptoms, and is leading a normal life.

Case 2.—A woman of twenty-six with extensive signs in the right lung, tubercle bacilli in the sputum, temperature 101 F., and signs of toxæmia was being treated by artificial pneumothorax. During the fifth refill, when 300 c.cm. of air had been introduced and the intrathoracic pressure was — 6 — 1, she suddenly became very cyanosed, gasped for breath and lost consciousness. She recovered in half an hour and afterwards began to improve. Fig. 13 shows the temperature. In this case the improvement did not last long. She relapsed and died about a year later.

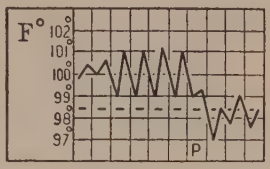


Fig. 13.—Temperature chart showing the effect of pleural shock occurring at P.

Case 3.—A man had a tuberculous sinus from the transverse process of one of the vertebræ. This showed no sign of healing, in spite of prolonged treatment. He was having mild sunlight treatment, and one day, after an exposure to the sun, which was more than double the usual, he had a reaction and high temperature, and felt that he had done himself harm. But the sinus began to heal after this, and has shown no sign of breaking down since.

Fig. 14 shows the temperature of a patient following only 10 c.cm. of serum. He never rallied and died in a few weeks. This is no isolated case, and a reaction, whether specific or not, may lead to disastrous results. For this reason most physicians try to avoid reactions, whatever drug or vaccine they may be using. In some of the cases I have described a violent reaction occurred, and it does not seem to me to be justifiable to cause such a reaction intentionally. The two

most dreaded and fatal accidents in pneumothorax treatment are pleural shock and rupture of lung, yet in Case 2 the patient had a definite period of improvement after pleural shock, and in Case 1 the patient suddenly began to improve and made an uninterrupted recovery after reaction resulting from rupture of the lung.

It is not only in cases of tuberculosis that improvement may follow shock. During the war I treated several cases of enteric fever with intravenous injections of colloidal gold. The injection was generally followed by a rigor, or at least a sudden rise in the temperature, but when the initial reaction was over the temperature would fall for a few days, and in some cases would remain normal. The same may be seen when mercuric chloride is given intravenously in cases of toxic pneumonia or other acute infections. It is difficult to believe that so small a dose of mercuric chloride as gr. $\frac{1}{16}$ can have any direct effect on the micro-organisms, and it would appear that the result is due to the shock.

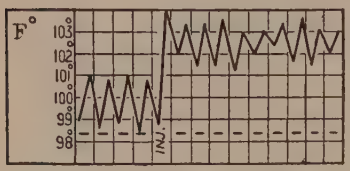


FIG. 14.—Chart showing the effect of an intra-muscular injection of 10 c.cm. serum.

For these reasons when a patient with pulmonary tuberculosis is rapidly or steadily losing ground and other treatment has failed, I think it is good treatment to increase the dose of sanocrysin, serum, or whatever is being given until a reaction is produced. The most striking case of improvement from this method occurs in young patients of fourteen to eighteen years of age with acute pulmonary tuberculosis. It is, however, a method that must be used with the greatest caution, and one should always start with small doses to avoid a severe reaction in a patient especially sensitive.

REFERENCE

(1) BURRELL, L. S. T. "Shock Tactics in Pulmonary Tuberculosis," *Lancet*, 1927, *i.*, 1212.

Calcium.—Calcium has been advocated in the treatment of tuberculosis, especially to control hæmoptysis or diarrhœa. Prest (1) describes a series of cases treated with collosol calcium. He gave 0·5 c.cm. hypodermically at intervals of a week or longer. In a few cases there was some reaction, but no serious complication arose. In many patients he noted distinct improvement, such symptoms of activity as night sweats disappearing readily under the treatment.

Ringer and Minor (2) describe the treatment of intestinal tuberculosis by injections of 5 to 10 c.cm. of a 5 per cent. solution of calcium chloride. They formed the opinion that this treatment gives great relief in cases of tuberculous diarrhœa. When there is actual tuberculous ulceration of the intestine it is difficult to understand how calcium can influence the condition, for post-mortem examinations show that the ulceration remains even after prolonged calcium treatment. However, as these authors point out, a drug which does good, though we know not how, is of more value than one which fails to relieve, although in theory it should do so.

In my experience calcium given by any method has no definite effect on tuberculous diarrhœa. I have seen improvement, but have never proved that this was in fact due to the calcium. This type of diarrhœa can usually be controlled by diet, and if milk is withheld and a more solid diet substituted, it is almost always much relieved if not quite stopped.

Maendl (3) describes the results of treating 250 cases with intravenous injections of calcium, and he notes improvement and disappearance of symptoms of activity. For hæmoptysis he gave 5 c.cm. of a 10 per cent. solution of calcium chloride intravenously, and repeated the injection eight-hourly. The result of this treatment was very satisfactory, and the hæmorrhage sometimes stopped with remarkable rapidity. H. Elving (4) also found calcium chloride a successful hæmostatic. He gave as much as

3 grm. (20 c.cm. of 15 per cent. solution) of calcium chloride intravenously without ill-effect, and found that the coagulation rate of the blood in one case was increased from five and a half to half a minute, and there was always a considerable increase.

I have given weekly subcutaneous injections of 1 c.cm. collosol calcium to thirty sanatorium cases, and did not observe any change which could be attributed to the treatment. Most of the patients improved, as most do in a sanatorium, but it was impossible to say from the symptoms or course of the disease which patient had calcium and which had not.

Given intravenously I have seen no effect except an occasional slight reaction such as so frequently accompanies other drugs injected intravenously. I have tried daily intravenous doses of 5 c.cm. of a 10 per cent. solution in acute cases without any result.

For hæmoptysis calcium may well be tried. It will not, of course, control a large hæmorrhage, but it may check continuous slight bleeding, and, moreover, it is a perfectly harmless method of treatment.

REFERENCES

(1) Prest, E. E. *B.M.J.*, 1922, *i.*, 53, and *ii.*, 283.
(2) Ringer, P. H., and Minor, C. L. *Am. Rev. Tub.*, 1922, **V.**, 876.
(3) Maendl, H. *Zeitsch. f. tuberk.*, 1921, **XXXV.**, 184.
(4) Elving, H. *Abstract Tubercle*, 1922, **VII.**, 323.

Sodium Morrhuate.—This is a sodium salt of cod-liver oil, which has been advocated in the treatment of pulmonary tuberculosis. It is given by subcutaneous injection in doses of from 0·1 to 1 c.cm. of a 3 per cent. solution. In forty cases treated at the Brompton hospital with doses of 1 c.cm. twice a week, I found no benefit, and there was no evidence of any more gain in weight than occurred in similar patients not having the treatment. I have frequently given a course of injections to out-patients who were losing weight or doing badly, and have found it without effect.

Dr. Jessel (1) treated seventeen male patients at Peel Hall Pulmonary Hospital, and his conclusions were " no evidence that subcutaneous injections of a 3 per cent. solution of sodium morrhuate in doses of 0·1 to 0·9 c.cm. have any material influence upon the course of tuberculosis in the chronic or advanced stages."

REFERENCE

(1) Jessel, G. *Tubercle*, 1925, **VI.**, 223.

CHAPTER IX

TREATMENT—*Contd.*

Sanatorium Treatment

Sanatorium treatment consists of many factors of varying importance to the combined action of which results are due. It may be said to have developed out of the "open air therapy," but it must be clearly understood that fresh air is but a small part of the treatment, and so-called "sanatorium treatment at home" usually fails because it is not possible to produce at home all the factors which are present in a sanatorium. Sir J. Kingston Fowler (1), said: "The so-called 'open air' method was first systematised and carried out in a sanatorium, and although the name is not free from objections, I think it is better to call it the 'Sanatorium treatment'; we shall at any rate, by so doing, avoid the risk of its being supposed that a patient with tuberculosis of the lungs, who is merely being exposed to the open air, is being treated according to any approved method." It may be said that there are two main factors in sanatorium treatment: First, to teach the patient how to live under his altered circumstances; and, secondly, to train the body to as high a condition of physical fitness as possible. Of the many factors which go to make up this treatment may be mentioned rest, exercise, fresh air, diet, routine life with regular habits, treatment of symptoms, etc. This system of treatment cannot be followed out except under constant supervision, and the degree of its success is largely dependent on the personality of the medical superintendent. Nordrach-in-Baden Sanatorium, in the Black Forest, was founded by Otto Walther, a pioneer of sanatorium treat-

ment. It was said of him that he inspired the patients with courage and the desire to live and to work. The same may be said of Trudeau, who started the sanatorium at Saranac Lake. Those who say that sanatorium treatment has failed are ignorant of the state of things before it was introduced. They often fail to understand the type of case for which a sanatorium is intended or are unlucky or unwise in their choice of a sanatorium. The site of a sanatorium is important, but of far greater importance is the medical staff, especially the medical superintendent.

It is impossible to over-rate the value of sanatorium treatment at the proper stage of the disease, and although very few patients want to go into a sanatorium the great majority of them settle down when there and they will admit that they are getting treatment and management which cannot be obtained at home. Very occasionally one finds a patient whose temperament is such that he finds the routine too irksome and has to leave, but this type of patient almost always does badly. Most patients, when once they have become accustomed to sanatorium ways, prefer to stay as long as their treatment requires many restrictions. To them life as an invalid at home, with brothers and sisters leading a normal life around them, is harder than at a sanatorium with others leading a similarly restricted life.

Dr. Wingfield (2) gives three main objectives in sanatorium treatment :—

1. Regaining health or resistance.
2. Consolidating this gain.
3. Education.

He points out that the best results can be obtained only after long treatment, such as nine months or a year, although in more advanced cases, where arrest is out of the question, patching up can be accomplished in half or quarter of this time. Those who have visited Swiss sanatoria must have been impressed with the length of stay in the sanatorium of

some of the most successful cases. It is no uncommon thing to find a patient who has been two or more years in the institution. The co-operation of the patient is essential, and Dr. Wingfield says that a patient should be encouraged to ask, " How well can I get " rather than " How soon can I get home." He also emphasises the fact that treatment should be varied according to the patient and should not be rigidly the same for all. " What is grade 5 to an ill-developed, elderly, emphysematous clerk may be only grade 2 to a muscular young policeman."

Rest is an important part of sanatorium life and is taken lying on a bed or couch at stated hours, such as 12 to 1, 2 to 4 and 6 to 7. In most Swiss sanatoria these rest hours are called " cures," and are usually spent on couches on the balcony. The hours of getting up and going to bed, and the exact amount of rest taken during the day, will depend on the condition of the patient. Exercise is also necessary, and one of the most difficult parts of sanatorium treatment is to judge the correct proportion of rest and exercise required to give the best results.

At many sanatoria walking is the only form of exercise permitted, and it is a very good rule to allow no other form of exercise for a least two years after the disease has become arrested. Patients should never run and should not walk quicker than three miles an hour. Relapse is more likely to occur from over-exercise, especially outdoor games, than from any other cause.

For patients who have to earn their living by manual labour a system of graduated labour may be employed. Marcus Paterson (3) developed this system at Frimley Sanatorium.

It is well known that exercise is followed by a reaction in a tuberculous patient, and Paterson believed that these reactions, if carefully controlled by the physician ordering the right amount of exercise, were beneficial and that the patient was in effect being treated by his own tuberculin.

Whether this is the true explanation or not, the results of the treatment were very good, and many patients left the sanatorium capable of doing a full day's manual labour without harm. At the present time a system of graduated labour is employed at Frimley Sanatorium. The patients at first keep to walking exercise, but when they are sufficiently strong they begin a system of grade work.

Grade 1.—Carrying small gardening basket about three miles.

Grade 2.—Carrying heavier basket the same distance. Light painting. Potting.

Grade 3.—Hoeing and light digging. Using light roller or hand cart.

Grade 4.—Digging and trenching with small spades. Cross-cut sawing.

Grade 5.—Digging and trenching with full-sized tools. Garden truck work.

No rapid work should be allowed and sudden spurts are harmful. It is most helpful to have an ex-patient as foreman to supervise the working parties, for this gives great encouragement to the patients, who see in the "cured" foreman an example of the results of treatment. There is undoubtedly more danger in employing graduated labour than merely walking exercises, and the work must be supervised with the greatest care, but amongst the working classes it has many advantages.

Drs. Cooper and Dow (4) describe certain exercise tests which were employed at Frimley Sanatorium. They took 100 patients, and those who seemed likely to respond well to treatment and to have a good ultimate prognosis they classed as satisfactory, the others as unsatisfactory. There were forty satisfactory and sixty unsatisfactory in their series. The tests consisted in the effects of exercise (such as a four-mile walk) on pulse, temperature and respiration. They classified the results of these tests as :—

Good.—If the pulse-rate fell to within ten beats of the

resting pulse-rate within five minutes of stopping exercise and there was no dyspnœa or rise of temperature.

Intermediate.—If the pulse-rate fell to within ten beats of the resting pulse-rate within half an hour of stopping exercise and there was no rise of temperature and no marked dyspnœa.

Bad.—If the pulse-rate did not fall to within ten beats of the resting pulse-rate within half an hour. There may be a rise of temperature and dyspnœa.

The results of these tests were, in the forty satisfactory cases :—

Good, 32·5 per cent. Intermediate, 62·5 per cent. Bad, 5 per cent.

In the sixty unsatisfactory cases the results were :—

Good, 18·3 per cent. Intermediate 31·6 per cent. Bad, 50 per cent.

A slight rise of temperature after exercise is not uncommon, but it should fall to normal in ten minutes. Temperature is the most important guide to the patient's capacity for exercise, but other signs, such as loss of weight, increase of symptoms, greater sedimentation rate or falling off of vital capacity, would suggest that a re-adjustment of treatment should be considered and usually indicate that more rest is required.

Diet.—The principles of diet have already been discussed. Dr. Wingfield (2) is of opinion that the normal weight in health for an individual need not be exceeded and that all attempts at stuffing or overfattening should be avoided. None the less, food is an important part of treatment, and one of the functions of sanatorium treatment is to educate the patients to take regular and sufficient, though not excessive, food. To assist digestion the teeth should always be examined and treated if necessary.

Milk is often regarded as essential ; an average of 1½ pints per patient daily, ½ pint being taken with porridge for breakfast, ½ pint at the midday meal and ½ pint in the evening

may be taken as a typical sanatorium allowance. In addition, milk is generously employed for puddings, custards, etc.

At Frimley less milk is given, and patients are kept as far as possible on a diet which they can afford to continue when they have returned home. Extra milk or other diet is of course ordered in special cases when required, but a patient who can keep fit only under a diet and mode of living which he cannot afford at home will tend to breakdown on leaving the sanatorium. Frimley is essentially a working-class sanatorium, and its chief object is to restore to the patient as much capacity for work as possible under conditions which he can maintain at home.

Cod-liver oil was often given as routine treatment, and it is undoubtedly a very good food and useful tonic, but it is not necessary in the majority of cases and it is best not to make a patient dependent on it. When a patient has lost much weight and wants special aid to regain it, or when the digestion is bad or some other reason is present, extra milk, cod liver oil, alcohol or tonics may be helpful, or even essential, but one should never forget that the best cases are those who can do without such extras.

Fresh Air.—Sanatorium treatment has developed out of the open-air theory of treatment, and fresh air is undoubtedly a very important factor in sanatorium life. I must repeat, however, that the object of an open-air life is to tone up the body and improve the general health and not to treat tuberculosis. In most cases it is easy to acquire the fresh air habit, and it has several advantages, the chief of which are a lessened liability to catch cold and an improved standard of general health. People who work in the open air but spend their evenings or leisure hours in stuffy atmospheres are often robust as a result of their work, but the changes from hot to cold temperatures are harmful, and bronchitis, pneumonia and colds are not uncommon amongst them. However, the comfort of warm rooms is such that most people are prepared to risk the various catarrhal conditions and

prefer a few colds to living a fresh air life. For the consumptive, however, the risk is much greater. A little bronchitis or even a bad cold in the head so frequently leads to renewed activity in the tuberculous lesion that it is essential for the patient to take all precautions against such catarrhal conditions. The patients and staff in a sanatorium very rarely catch cold, and after a time one does not feel the cold. I have frequently heard a visitor to a sanatorium complain of cold; a common saying is, "I like fresh air, but cannot stand draughts, they always give me a cold." Perhaps they do, but as it is impossible to enjoy life without meeting an occasional draught, why not get used to them? Of course there are degrees of cold, and it is quite wrong to expose consumptives or any one to all weathers; but overdressing, overheated rooms and screening off the slightest current of air do tend to make an individual soft and liable to catarrh.

It may be said that work in the open air is good for the general health and that, apart from this, a fresh air life (by which I mean open windows and the avoidance of stuffy atmospheres) is the best way to keep free from cold and other catarrhal conditions. With regard to open-air work, however, it should be remembered that most outdoor jobs mean manual labour and only a few consumptives can stand this. A delicate city clerk who, after leaving the sanatorium, gave up his office work and got employment on a farm would almost certainly break down with active tuberculosis, whereas if he returned to his office the disease might remain arrested permanently. If one bears in mind that there are people who are not physically fit for outdoor work, one will avoid the mistake of advising it for all consumptives, regardless of their condition, but simply because they are tuberculous. With regard to a fresh air life, one should also remember that there are people who cannot stand it. The average young patient will quickly get acclimatised to open windows and draughts and will be all the better for it. Elderly patients,

those who are very thin or delicate or have bad circulations, usually have to be protected from cold. It is a big mistake to expose such people to cold whatever the state of their tuberculosis may be, for they will do much better in warm rooms or in a warm climate.

Recreation.—It is very important to provide as much recreation as possible, but outdoor games are more responsible for relapses than any other cause. All energetic games, such as hockey, tennis, football, cricket, must be forbidden, and even golf is too much for most patients. Clock golf, putting, croquet, etc., may be allowed for many patients, and such games may be used in treatment in place of gardening or other forms of graduated labour. With regard to indoor games, each patient should be watched and should not be allowed to play a game which unduly excites him. Chess, played in the evening, may lead to a sleepless night, and I have known a patient's pulse go from 76 to 140 at an exciting moment in a game. In the same way many patients get excited at cards, especially if they are playing for more money than they are prepared to lose. If the patients are watched it is easy to detect any harm that may follow a particular game, but as far as possible the patients should be allowed to chose their own recreations, and all harmless amusements should be encouraged.

Routine.—A regular life is an essential part of the treatment, and the following may be taken as a typical routine :—

8.30.—Breakfast.

9.30—12.—Rest, exercise or recreation according to medical orders.

12—1.—Rest in recumbent position on balcony.

1.15.—Luncheon.

2—4.—Rest in recumbent position on balcony.

4.15.—Tea.

5—6.—Recreation.

6—7.—Rest in recumbent position.

7.15.—Dinner.

8—9.30.—Recreation.

9.30.—Bed.

This routine must, of course, be varied according to the seasons or the condition of the patient. For example, some will require more rest and others will be able to dispense with one or more of the rest-hours and go for walks or play games. Smoking should be allowed, for although, like everything else, it is harmful in excess, it is a great comfort to many people, and to deprive them of it only adds to their unhappiness and leads to discontent. In some cases it brings on cough and sputum, and it will often be found that sputum disappears when a patient gives up smoking. In such cases it should be stopped, but in the great majority of cases it will be found beneficial rather than harmful.

Sometimes, especially after prolonged sanatorium treatment, a patient becomes hypochondriacal and can think or talk of little else but his temperature and symptoms. This is not unnatural in such a chronic disease, but the physician must look out for the first signs of such a condition, as it is apt to leave the patient a mental wreck long after the tuberculosis is arrested. A change of environment or a holiday away from the sanatorium for a few weeks is often enough to restore the patient's balance, but if he is well enough to work this should be advised as soon as he shows the slightest sign of becoming hypochondriacal. Indeed, it is lack of occupation more than anything that causes such a condition. More consumptives relapse from play than from work, and one can frequently see a great improvement in a patient as soon as he gets settled in some suitable employment.

It is sometimes found that a patient who keeps perfectly well and can do a full day's work in a sanatorium relapses as soon as he leaves and returns to his previous life and occupation. Such patients do very well if they can obtain work in the institution, and in every sanatorium are to be found ex-patients keeping fit and doing hard work as doctors, nurses, clerks, porters, etc. Unfortunately there are not

enough sanatorium jobs for all these patients, who form a big group, and it is for this group that an after-care colony is especially useful. Papworth Village Settlement was started chiefly for this type of patient, although there is a hospital side in the settlement and all stages of pulmonary tuberculosis are treated. It is a growing village, and the sales from the industries, which were under £4,000 in 1919, exceeded £50,000 in 1927. The industries include leather-work, trunk-making, bookbinding, upholstering, furniture work, etc., and although the life is not suitable for all classes it undoubtedly fills a want and enables a number of patients to keep their health and, at the same time, to earn a modest living. After twelve years' experience not one of the children brought up in the settlement has developed any form of clinical tuberculosis.

REFERENCES.

(1) Fowler, J. Kingston. "Open Air Treatment of Tuberculosis," *Med. Chir. Trans.*, **LXXXIII.**, p. 1.

(2) Wingfield, R. C. "Modern Methods in the Diagnosis and Treatment of Pulmonary Tuberculosis." London, 1924, Chap. V.

(3) Paterson, Marcus. "Autoinoculation in Pulmonary Tuberculosis," London, 1911.

(4) Cooper, A., and Dow, D. *Lancet*, 1928, *ii.*, p. 859.

CHAPTER X

TREATMENT—*Contd.*

Climate.—A great deal of harm has been done by the failure to appreciate that the best climate for a patient varies according to the individual and to the stage and degree of his disease. A patient will often have a fixed belief that if he goes to a certain place he will be cured. One will want South Africa, another New Zealand, another Switzerland. One patient said to me, "They have found T.B. in my sputum, and surely that spells Switzerland for those who can afford it; all I want to know is, what part of Switzerland is best?" The truth is there is no place that is good for every consumptive, and how a patient lives is far more important than where he lives. Paterson (1) wrote: "Climate, provided it is not actually unsuitable, has little if anything to do with treatment."

Dr. J. A. Miller (2), discussing climate in the treatment of pulmonary tuberculosis, points out that there is no universally ideal climate. Selection of a suitable locality, he says, is "an individual problem for every patient, depending upon his temperament, tastes and individual reaction to environment as well as the character of his disease." He also says that contentment and reasonable comfort are essential, and that the patient must be able to afford to stay a sufficient time and have the necessary medical supervision.

A change of environment has for centuries been considered beneficial. Celsus (25 B.C.) wrote (the patient must): "Change his climate, taking care to remove to a grosser one than that he leaves, and therefore from Italy to Alexandria is a very agreeable change."

Roughly it may be said that acute cases are better at home in some institution where they can be under medical supervision, and any complication or development in the disease can have the appropriate treatment. There should be good ventilation with an abundance of fresh air, but it is not necessary for the patient to be uncomfortably cold, and some patients, especially those who are thin or have poor circulation, may suffer from too much exposure to fresh air. In this acute stage it is usually a mistake to send the patient abroad, and he should certainly not be sent to a high altitude. Dr. Miller (2) says: "Any change of climate involving the fatigue of travel is contra-indicated in acute cases with fever or hæmorrhage, or in the very far advanced and markedly debilitated cases."

During the subacute stage there is an advantage in sending the patient to a healthy place, and, as has already been said, a sanatorium is indicated at this stage. I think it is best not to send these patients abroad. The choice of an English sanatorium should depend on the patient, and, on general principles, a robust type of patient usually does best in a bracing climate, whereas a delicate one may get on better in some warmer place in the South or West of England. If a patient finds that a certain locality does not suit him he should be moved.

In the chronic or after-care stage the choice of climate is usually a matter of the patient's financial position. Many who have to work get on quite well, even in the big cities, and one must remember that there are certain advantages in living in a city. Light work in the country—for example secretary to a golf club in a health resort—is ideal; but such jobs are difficult to find, and country work usually entails hard manual labour, which, above all, is bad for this type of patient. A city clerk who is used to a regular sedentary life is far less likely to break down in a city office than if he went to work on a farm in the country, though if he had the offer of transfer to some branch office in the country he would be wise to accept.

For those who do no work I should advise gravel soil in a place protected as far as possible from rain-bearing winds. The exact place must depend on the patient individually, and I always advise him to stay there for a few months to see if it suits him before taking a house.

Many patients like to leave home for the winter, and, in England, Falmouth or Torquay are suitable for those with extensive fibrosis, or whose condition is complicated with bronchitis. For the robust patient who can take plenty of walking exercise St. Leonards or the East Coast is more suitable, and for the intermediate type of case Bournemouth or the Isle of Wight are to be preferred.

Foreign resorts are more helpful in this than in any other stage of the disease.

Switzerland.—The climate of the Swiss Alps is most suitable for those who have just finished the subacute or sanatorium stage. Provided a patient is well enough to take regular walking exercise and is moderately robust, he may derive great benefit from spending a winter in the Alps. The choice of a Swiss resort or sanatorium is a matter of great importance, as they vary enormously in efficiency and routine, so that a patient should never be sent to one unless it is known to be satisfactory. The best months for these resorts are November to April, and it is a mistake for a patient to return to England before the winter is over. One knows the great benefit which people derive from a winter holiday in the Alps, and, of course, the consumptive gets the same benefit, provided he is strong enough to take advantage of the facilities offered. Many patients spend the summer also in the Alps, but this has not the same advantage as the winter, when the climate is certainly fascinating and ideal for the right type of case.

Sir J. Kingston Fowler (3) writes : " As a rule, the patients who obtain most benefit from the change (*i.e.*, to the Swiss Alps) are young subjects who are fond of outdoor exercises, such as skating, and are strong enough to take part in them,

who, whilst in the mountains, spend the greater part of the day in the open air, and who are determined to do all in their power to regain their health." He gives the following contra-indications :—

" 1. The presence of certain complications, such as albuminuria, advanced disease of the larynx, valvular disease of the heart and extensive emphysema.

" 2. Extreme emaciation.

" 3. An irritable state of the nervous system with constantly quickened pulse and a liability to feverish attacks.

" 4. A decided inability to bear cold.

" 5. To these must necessarily be added those conditions which, as already stated, render a case unsuitable for climatic treatment abroad."

In this last group he would include patients with advanced disease, who, he says, should " only be allowed to travel to places at a moderate distance."

There is undoubtedly more temptation in the average foreign institution than in an English one, and, although a patient can obey rules and live a suitable life, it is easy for him to live unwisely. The disease is a very long and irksome one, and a number of young patients living together are apt to get into bad habits, especially if, as so often happens, they feel well in themselves, and believe that because they are in Switzerland and " taking the cure," no other treatment or precaution is necessary. For this reason it is important to choose the institution from personal knowledge, and also to consider the temperament and character of the patient. Another danger is that a patient may get a habit, so that he cannot, or thinks he cannot, winter anywhere except in Switzerland. This does not matter for a few well-to-do patients, but for the majority it is impossible. In general terms one may say that :—

1. Acute or advanced cases usually are harmed by treatment in high altitudes.

2. Subacute or sanatorium stage cases get no special advantage, but may do well.

3. Chronic cases whose disease is nearly arrested may get enormous benefit from a winter in Switzerland. It is an excellent method of putting the finishing touches to a cure.

French and Italian Riviera.—Patients with chronic pulmonary tuberculosis do not, as a rule, find the Riviera suitable. There are undoubtedly many days of bright warm sunshine, when the patient can spend most of the day out of doors, but it becomes very chilly when the sun goes down, and catarrhal conditions are common. If a patient has a villa where he can have home comforts he may do very well through the winter on the Riviera, but in the majority of cases it is better to recommend some other place for the winter.

Egypt and the North Coast of Africa.—Here the atmosphere is dry and pure, and there is an abundance of sunshine. Some places, however, are dusty, and there is a great difference between the day and night temperature. There are many places on the North Coast of Africa which are much preferable to those on the other side of the Mediterranean, but the physician would be well advised to recommend only those places that he knows from personal experience.

Canary Islands.—In these islands the climate is as nearly perfect as possible for many cases, and the only disadvantages are that to get to them necessitates a sea voyage, and life there is somewhat dull. This latter reason may not always be a disadvantage.

Dr. Lucas (4) has written an interesting description of the islands, and he gives the table on the following page, showing the meteorological records from Las Palmas (Grand Canary) during the months of October to May.

It will be seen how very equable is the climate. The variations between the day and night temperature is never more than 10°. At Orotava (Teneriffe) the rainfall is slightly higher and the sunshine less than at Las Palmas,

	Oct.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.
Mean shade temperature	71·4	67·6	64·7	62·3	63·9	63·0	64·6	66·5
Surface sea temperature	72·4	70·1	67·5	65·9	65·1	65·4	65·9	67·3
Amount of rain in inches .	1·06	1·75	1·57	1·68	0·57	0·79	0·40	0·55
Sunshine in hours . .	189	165	161	168	183	189	190	218

Meteorological records, Las Palmas, Grand Canary.

and this is said to be due to the elaborate system of irrigation in connection with the banana industry which requires a generous supply. At Las Palmas is a British hospital with private rooms available for visitors, and staffed by English nurses. There is also a good social club, a lawn tennis club and an eighteen-hole golf course.

Distant Health Resorts.—There are many excellent places in Australia, New Zealand, S. Africa, America, etc., but none of them present any special advantages which cannot be obtained nearer home. I cannot discuss them in this book, but may mention that most of them have bad as well as good seasons. Some of the country round Bloemfontein, for example, is ideal during certain months, but during others is either a desert of dust or a bed of mud. To visit such places during the proper season may be good, but to take a farm and settle down is usually unwise. Again, many distant places that sound attractive are suitable only for those who can do hard manual work, and are quite impossible for the tuberculous patient who has to earn his living.

Sea Voyages.—For a patient in fair general health who wishes to avoid the English winter a sea voyage around S. America, S. Africa, Australia, or some such trip south, may be a very healthy way of spending the winter. The good food, the rest, and the opportunity for fresh air and sunshine are all satisfactory.

In the treatment of active tuberculosis, however, a sea

voyage is almost always a mistake, and more likely to do harm than good, quite apart from the fact that the patient may not be allowed on the ship or to land at certain ports of call.

In conclusion it may be said that climate is of secondary importance in the great majority of cases of pulmonary tuberculosis. People get accustomed to their own home conditions, and it is usually a mistake to send them for long periods to a totally different climate if, after treatment, they have to live at home again.

The ideal spot for a consumptive to live might be some country district where there are no extremes of heat or cold in summer and winter, where there is protection from rain-bearing winds and the house is on gravel soil. But even under such conditions it is doubtful whether the patient would have a longer expectation of life than one who chose his home to suit his own happiness and his business and social convenience.

For the consumptive the important question is, " How shall I live ? " not " Where shall I live ? "

REFERENCES

(1) Paterson, Marcus. " The Shibboleths of Tuberculosis," London, 1920, p. 225.

(2) Miller, J. A. " Climate in Pulmonary Tuberculosis," *Tubercle*, 1929, **X.**, 217.

(3) Fowler, Sir J. Kingston. " Pulmonary Tuberculosis," London, 1921, p. 218.

(4) Lucas, Geoffrey. *Post Grad. Med. Journal*, 1928, **IV.**, 37.

Phototherapy.—Dr. Crocket (1), discussing ultra-violet ray treatment in tuberculosis, says that rays with therapeutic properties may be divided into four classes, all electro-magnetic and differing only in the length of wave :—

1. Those from radio-active substances, such as radium. Here the rays are only from 0·15 to 0·015 Angström units. There are three kinds, called alpha, beta and gamma. The alpha rays do not penetrate, the beta have slight, and the gamma considerable penetrating power.

2. Röntgen rays range from 0·15 to 12 Angström units, and have many gamma rays with great penetration.

3. The so-called ultra-violet rays measure from 2,100 to 3,800 units. They lie between the luminous spectrum and the area of Schumann and Lyman. Strictly speaking, of course, all rays beyond the violet in the visible spectrum are ultra-violet, which therefore include X-rays and radium.

4. Rays of the visible spectrum. These are from 3,800 to 7,600 units. The violet end of the spectrum has considerable chemical but little heating action, and the red end has great heating but no chemical powers.

The sun has ultra-violet rays, but none shorter than 2,900 Angström units, as the shorter ones are filtered out by the smoke and moisture in the atmosphere. The purer the air, therefore, the more ultra-violet rays are present, so that the pure rarefied air of a mountain may contain twice the amount of ultra-violet rays as the atmosphere of a valley. The short rays do not penetrate as much as the longer ones, but they are more irritating and bactericidal and are filtered out before the longer rays. Water and snow reflect the rays, and increase their power considerably, and for this reason people get " sunburnt " so readily at the seaside. It is not the warmth of the sun, but the chemical action of the violet rays which produces sunburn. The pigment protects against the ultra-violet rays, so that when well sunburnt one can stand much exposure to the sun without reaction.

The effect of the rays is also increased by the movement

of the air, and hence they are not so effective if given artificially in a room where the air is still and warm. Professor Leonard Hill (2) emphasises the importance of cool air passing over the skin if it is desired to get any therapeutic effect from sunlight or artificial rays. With actual sunlight there are other rays which assist the therapeutic action of the ultra-violet ones.

Dr. Crocket (1) describes the following direct effects of ultra-violet therapy :—

1. Vasodilatation and œdema of the soft tissues exposed to the light. He points out that this relieves congestion in internal organs, and no doubt accounts for the improvement in the condition of the skin and the growth of hair and nails, which are so often noted in those exposed to ultra-violet rays.

2. Sterilisation of superficial tissues. The short ultra-violet rays are strongly bactericidal, and as these are filtered out by impurities in the atmosphere before the long ones, the rays of the sun have much more bactericidal effect at high altitudes than at low ones, especially low altitudes, where there are much smoke, moisture and other impurities in the air.

3. Relief of pain and discomfort. This is especially noted if there is an excess of the short, and therefore more irritating, rays, and consequently for this purpose also treatment at a high altitude will be more effective than at a low one.

4. Pigmentation. He describes two forms of skin reaction, an immediate transitory reddening which disappears half an hour after exposure, and an erythema which appears from six to ten hours after exposure, and which may go on to severe desquamation and blistering if the exposure has been too long. This " sunburn " is produced only by rays below 3,200 units. He says that although this pigmentation is protective, it changes a white reflecting surface into a dark light absorbing one, and therefore favours the absorption of rays.

The rays also have an indirect effect, and are said to

affect not only the general health, but also to cause alteration in the blood chemistry.

Dr. Spence (3) made an investigation of twelve adult tuberculous patients; of these eight had pulmonary disease with tubercle bacilli in the sputum. Irradiation was given by a Hanan mercury vapour lamp of 40 units power. It was found that the blood calcium and phosphate were not affected by the light. The blood cholesterol, however, was raised, the greatest rise being seen in the patients who showed the greatest improvement clinically. In one case after ten exposures the patient had a febrile reaction and vomited. His blood was examined, and no change was found in the calcium or phosphorus, but the cholesterol had fallen from 160 to 140 mg. per 100 c.cm. of serum, and the irradiations were discontinued.

Dr. Crocket (1) has noted a definite improvement in the general health and mentality of the patient, and also a reduction of blood pressure when this was high. He has seen a fall of as much as 20 mm. Hg in the systolic pressure as a result of light treatment.

In certain cases of surgical tuberculosis treatment by light is undoubtedly of great value. Much depends, of course, on the way it is given and on the experience of the doctor in charge, but, provided it is properly administered, one will frequently see rapid improvement of certain conditions which are not amenable to other treatment, for example, tuberculous ulceration of the fauces.

In pulmonary tuberculosis, however, the value of light treatment is a matter of doubt. Some deny that it has any value at all, and certainly its value, if any, is not great. It may, moreover, do harm if carelessly employed. Many patients with tuberculosis of the lungs get on better in winter than summer, and any great improvement noted when fine weather comes is more likely to be the result of removing the feeling of depression which so often goes with gloomy weather than to any effect of the ultra-violet rays.

Direct exposure to sunlight or ultra-violet rays will, if overdone, produce a shock reaction in cases of pulmonary tuberculosis, and, although this may be harmful, it may actually be beneficial, and, as with shock from other causes (tuberculin, serum, vaccine, etc.), the patient's condition and temperature may rapidly improve after recovering from the initial reaction. The reaction following over-exposure to sun is very similar to that produced by tuberculin.

Dr. Mathieu (4) devotes a chapter to the effects of sunlight on pulmonary cases. He quotes the opinions of doctors of Leysin who have had experience of the treatment, and also the views given at the conference at Leysin in 1910. The general opinion was that no better results were obtained by light therapy than by the ordinary methods of treatment, but that properly employed it was a safe procedure. M. Malgat obtained good results at Nice, and it was generally noted that the general well-being of the patients was improved. Dr. Rollier (5) found that patients having pulmonary tuberculosis complicating some surgical lesion did better than those with uncomplicated pulmonary tuberculosis. He saw a patient with recurrent hæmoptysis who was cured by sun treatment tried as a last resort. Many physicians consider light treatment contra-indicated in pulmonary cases, and Mathieu (4) quotes Dr. Jaquerod, of Leysin, as describing the case of a patient who had never had hæmoptysis until he went for a sun cure to the Mediterranean coast. Dr. Jaquerod said that light treatment did not appear to modify the results obtained by ordinary means, and was not without danger. Dr. De Reynier, at the same meeting, said that although he had never seen any improvement in the local condition as shown by auscultation, and had not seen diminution of sputum or loss of tubercle bacilli, at the same time he had never met with hæmoptysis, febrile reaction or increase of symptoms, and thought further trial should be given to the treatment.

Dr. Burnand referred to the similarity which existed

between the reactions following over-exposure to the sun and those resulting from tuberculin injections. He admitted that the general stimulant and tonic effect of heliotherapy was unquestionable, but thought that its focal effect was less certain. Its action depends largely on the state of the lung, and in cases of great activity Dr. Burnand considered it unwise to start a form of treatment capable of causing rapid exaggeration of congestion already excessive.

Dr. Crocket (1) gives the following summary of his results from seventy-two cases of pulmonary tuberculosis treated with light treatment :—

1. The general condition and the mental outlook were improved in practically every case.

2. The lung lesions showed fewer indications of activity.

3. The cough was relieved in 92 per cent. of the cases. The sputum was much reduced or ceased in 54 per cent. of the cases and in 20 per cent. was unaffected.

4. Wheezing, often indicative of congestion, is frequently removed by light treatment.

5. The appetite was usually improved. Of the cases with anorexia or dyspepsia 76 per cent. received definite benefit. It is not advisable to give light treatment for one hour before meals, as patients usually complain that it interferes with their ability to enjoy their food.

6. Weight was not benefited as often as expected. Thirty per cent. of the cases did not increase in weight.

7. Sleep was often greatly improved, though in nine cases it was apparently affected unfavourably.

He says that of these seventy-two cases improvement occurred in 85 per cent., whereas, apart from light treatment, 67 per cent. of the patients in the same institution improved.

Hæmoptysis occurred in four cases in the series, but Dr. Crocket thinks that it would have occurred apart from the treatment, and certainly it does not seem unduly frequent. In ten cases which had been running an intermittent tempera-

ture for several months, this subsided during the treatment. He gives the following contra-indications :—

1. Cases of progressive disease.
2. Cases in which destruction of tissue is marked and progressive.
3. Cases with obvious cachexia and amyloid disease, or other toxic manifestations.
4. Cases of sepsis with pyrexia.
5. Cases of hyperthyroidism.

Dr. Trail, at Midhurst, found that the sedimentation rate improved as the patient's condition improved. In common with other observers he found a reduction in the sedimentation rate following such treatment as artificial pneumothorax or sanocrysin injections. He found no change in the sedimentation rate, however, resulting from light therapy.

Most of those who use sun or artificial light treatment try to avoid reactions. When a reaction does occur, its result is very similar to that following tuberculin, serum, etc. Apart from the effect of such a reaction or shock it may be said that light treatment in pulmonary tuberculosis probably has no direct action either for good or bad. It has, however, two indirect effects which may be of considerable benefit to the patient :—

1. Light, and especially a bright sunny day, has a stimulating and cheering effect on every one including the consumptive patient.
2. The mental effect of feeling something definite is being done, and of seeing the skin gradually becoming pigmented, added to the improved appetite and feeling of well-being which usually goes with bright weather may assist the patient's recovery to a very great extent.

The method by which light acts is not clearly understood. Some think that the action is specific, and it produces an auto-tuberculinisation. This view is supported by the undoubted fact that an over-exposure to the sun produces a reaction which is very similar to that following the use of

tuberculin, but one must not forget that the same result can be obtained by the intravenous injection of T.A.B. or other non-specific substances. Rollier suggests that the pigment transforms the short wave rays into the longer and more penetrating ones.

If light treatment is employed in pulmonary tuberculosis the actual sun is better than artificial light, for the other rays besides the ultra-violet found in sunlight, combined with the fresh air, play a large part in the effect. For artificial light treatment the tungsten arc lamp, or the quartz mercury vapour, are best for ultra-violet rays, the tungsten arc being especially rich in short-wave rays; the carbon arc has, in addition to the violet rays, a large proportion from the red end of the spectrum.

In order to avoid reactions it is best to start with a short exposure of a limb, and if this produces no reaction the length of exposure and amount of the body exposed is gradually increased day by day. When the patient is thoroughly bronzed it is possible for him to remain exposed to the direct rays of the sun for hours without getting a reaction.

REFERENCES

(1) Crocket, J. *Tubercle*, 1926, **VIII.**, 1.
(2) Hill, L. "Sunshine and Open Air," Arnold & Co., London, 1925.
(3) Spence, K. C. *Brit. Journ. Actinotherapy*, 1928, **III.**, 148.
(4) Mathieu, G. "Importance des Résultats obtenus par L'héliotherapie à l'altitude dans le Traitement de la Tuberculose." M. Colin. Nancy: 1922, p. 176.
(5) Rollier, A. "La cure de Soleil," Paris, 1914.

CHAPTER XI

TREATMENT—*Contd.*

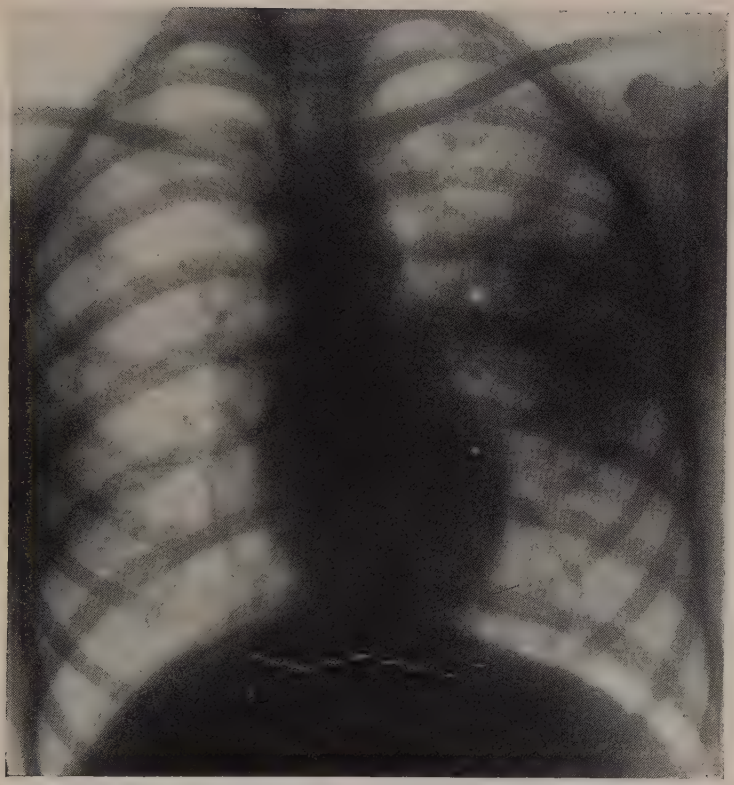
Artificial Pneumothorax

Many methods of treatment and so-called cures of pulmonary tuberculosis have been advocated, but very few have stood the test of time. Artificial pneumothorax was suggested on theoretical grounds more than a century ago and now is employed all over the civilised world. It is generally recognised by those with experience of tuberculosis that the basis of treatment is rest of the diseased part. The spongy consistency and continual movement of the lung make it easy for tuberculosis to spread, and it is not surprising therefore to see the rapid improvement which so often follows the induction of a pneumothorax.

The principle of the treatment is to produce lymph stasis and to rest the lung, thus preventing spread of the disease and allowing nature to effect a cure. As with every form of treatment there are enthusiasts who regard pneumothorax as the whole of the treatment, advocate it on any and every occasion, and think no other treatment necessary ; on the other hand there still remain a few die-hards who deny that it has any value at all. If one appreciates the purpose of the treatment it is possible to avoid either of these extreme views and to find in artificial pneumothorax one of the most valuable and effective weapons against pulmonary tuberculosis, and at the same time to recognise its limitations.

Selection of Cases.—In 1922 I gave (4) certain indications and contra-indications for artificial pneumothorax and now, although still in general agreement with them, I advise the

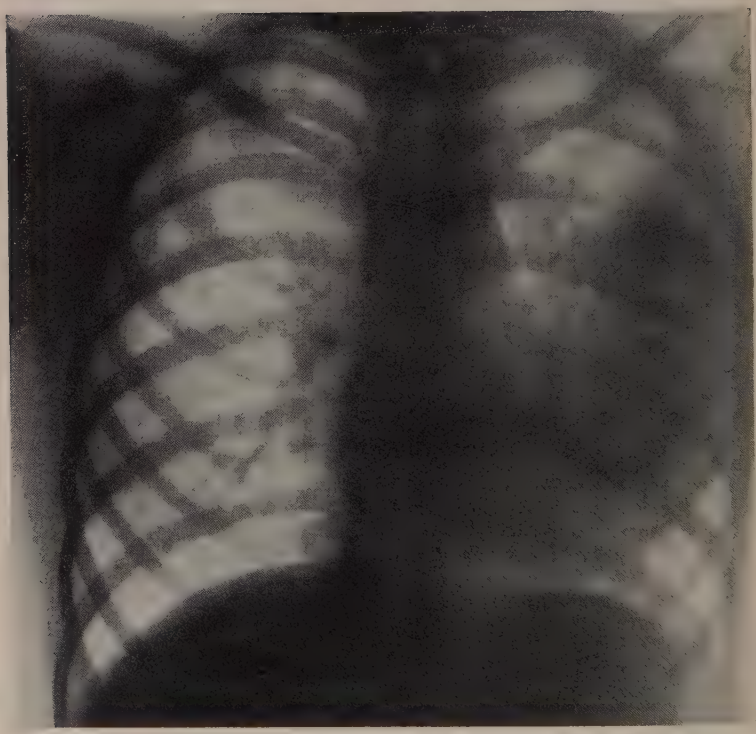
PLATE XXIII



Early Acute Tuberculosis of the Left Lung on January 12th, 1925.

[*To face p.* 134.

PLATE XXIV



The same Case as Plate XXIII taken on February 4th, 1925, showing Extensive Development in Three Weeks.

treatment more readily and am less inclined to consider certain contra-indications.

A. In all unilateral cases of pulmonary tuberculosis pneumothorax should be considered if :—

1. There are constant rales to be heard over an area corresponding to two ribs and intercostal spaces or more, with T.B. in the sputum and corroborating X-ray findings. In such a case, if the disease was of long standing, and the general condition or working capacity of the patient were good (*i.e.*, a chronic fibroid case with localised lesion), one might consider it unnecessary to collapse the lung, but in the majority of unilateral cases with such signs this treatment is indicated.

2. The disease is acute. In acute disease some of the signs are catarrhal, and clear up quickly with rest in bed, but the danger of spread to the other lung is so great, that in any acute febrile case with definite signs confined to one lung it is safer to induce a pneumothorax. Plate I shows the X-ray of a child on January 12th, 1925. She then had harsh breath sounds at the root of the left lung and a high intermittent temperature, but very slight cough and no tubercle bacilli were found on examination of the sputum. She was admitted into hospital on February 4th, 1925, and Plate II shows how the disease had spread. At this time crepitations were heard over the upper part of the left lung, and she had developed purulent sputum containing large numbers of tubercle bacilli. Her temperature was swinging between 98° F. and 103° F., but fortunately the condition remained unilateral. The lung was at once collapsed, after which the spread was stopped and she made a good recovery. Now January, 1929, she is alive and free from symptoms.

3. In spite of rest in bed the activity persists or improvement is not satisfactory. As a common example one may quote the patient whose condition does not become stable and the slightest exertion, such as getting up for two hours, causes a rise in temperature or some other symptom.

4. The patient has to earn his living or for some reason is unable to undergo prolonged treatment. There is no doubt that with the affected lung collapsed a patient is able to lead a more active life with much less risk of a breakdown than one not so treated.

5. There is repeated or severe hæmoptysis.

6. There are cavities with copious sputum.

7. There are certain complications, such as tuberculous laryngitis. If there is actual ulceration of the intestine pneumothorax is, of course, useless, but in many cases of diarrhœa or other gastro-intestinal symptoms resulting from toxæmia, the symptoms may be completely relieved when the diseased lung is collapsed.

B. In bilateral cases the indications for inducing artificial pneumothorax are affected by the following considerations:—

1. The disease is being treated in only one lung by unilateral collapse; if, therefore, there is any active disease in the other lung this also must be treated. In other words, the pneumothorax is affecting only part of the tuberculous lesion and not the whole as in a unilateral case.

2. If one lung is much worse than the other collapse may remove so much of the toxæmia and cause so great an improvement in the patient's general condition that the disease in the better lung also improves.

3. On the other hand collapse of one lung may increase the activity in the other. This is especially the case when there is early acute bilateral disease.

From this it follows that artificial pneumothorax may be considered under the same condition in bilateral as in unilateral cases of pulmonary tuberculosis provided one remembers that—

1. Treatment is directed on only part of the diseased tissue and the other parts also require treatment.

2. Collapse of one lung may aggravate the disease in the other.

In coming to a decision as to the wisdom of inducing

pneumothorax in a bilateral case it is more important to consider the degree of activity than the actual amount of disease, as determined by physical signs, in the better lung.

Bilateral Artificial Pneumothorax.—This may be done by two methods.

1. Producing partial collapse of both lungs at the same time.

2. Collapsing first one lung and after allowing it to re-expand, collapsing the other.

1. There is a large amount of reserve in lung tissue so that life can continue even after the greater part of the lung is destroyed. In most cases of bilateral disease there are adhesions which prevent the lung from collapsing sufficiently to endanger life, and it is remarkable how much collapse can be obtained in both lungs without any apparent ill-effect on the patient. There are, however, two great difficulties in producing successful pneumothoraces simultaneously on both sides. The first is that one cannot obtain complete collapse on both sides at the same time, and it is in cases of complete collapse that one obtains the best results. The second is that in acute cases, when there is considerable disease in both lungs bilateral collapse is apt to produce so much dyspnœa that the treatment has to be stopped, whereas in the more chronic cases with adhesions bilateral collapse is well borne, but usually unnecessary.

The type of case where the most striking results of collapse therapy are seen is the acute case with high fever and rapidly spreading disease, but remaining confined to one lung. This type of case is common in girls of about fourteen to eighteen years, and to a somewhat less extent in boys from about seventeen to twenty-one. Unfortunately, these cases are almost always bilateral, and in my experience simultaneous bilateral pneumothorax produces increasing dyspnœa and has to be stopped before the treatment has had any effect on the spread of the disease. It might be supposed that even a partial bilateral pneumothorax would give considerable

rest to both lungs and so tend to do good, but in practice this is not the case.

In more chronic bilateral cases a simultaneous collapse of both lungs may be tried, but the results are not promising, probably because it is possible to produce only a partial collapse. In unilateral cases the results of those where an efficient collapse is obtained, and those where the collapse is incomplete, are very different, and it is not surprising therefore to find disappointing results in bilateral cases where only partial pneumothorax is obtained. When, however, there is active disease in both lungs and the disease is slowly spreading, bilateral collapse may check the rate of spread and prolong the life of the patient. I always collapse one lung first and watch the effect on the other, which I should not collapse at the same time unless it showed considerable activity which could not be checked by other means.

Sometimes it happens that during pneumothorax treatment active disease appears in the non-treated lung and begins to spread rapidly. In such a case it is often necessary to collapse this lung without waiting for the other to re-expand.

2. I have obtained better results by collapsing first one lung and then the other, and where possible, I prefer this method to simultaneous bilateral collapse. In most cases of bilateral disease requiring pneumothorax the best that can be expected is to stop the rate of spread and leave the patient with chronic fibroid disease.

Bilateral collapse has been practised for many years, and Forlanini (5) had two cases which he published in 1911.

Burnand (6) induced a right artificial pneumothorax in a patient in June, 1915. In October, the patient had lost the tubercle bacilli from the sputum and was free from symptoms. In April, 1918, he had influenza followed by hæmoptysis and pyrexia. In July he had tuberculous laryngitis; signs of active disease appeared at the left apex, and tubercle bacilli were present in the sputum. The pneumothorax treatment was stopped and the right lung re-expanded. In January,

1919, a left artificial pneumothorax was induced carefully, a negative pressure being left after each refill. The patient was very dyspnœic, but was able to get up and walk about quietly. He died suddenly, in May, 1919.

Burnand's second case was a patient for whom a left artificial pneumothorax was induced in January, 1918. The patient improved at once, but the symptoms returned in October, and signs of active disease on the right side were found. The treatment was stopped, and the right lung became gradually worse until June, 1920, when a right pneumothorax was induced. The dosage varied from 200 to 500 c.cm., and the intervals between each injection were about eight days. A negative pressure was left after each refill. Sixteen refills were given. There was some improvement, but at the end of September right pleural effusion developed, and there was much dyspnœa. The heart became very weak, and the treatment was discontinued.

In one of my patients there were extensive signs of active disease over the left lung, and over the right lung as far as the third rib in the mid-clavicular line in front, and the angle of the scapula behind. She had just returned from a sanatorium, where she had become much worse, the physical signs having extended very rapidly. A left pneumothorax was induced, and four refills were given. The treatment was then stopped for seven weeks, when a right pneumothorax was made and five refills given. The patient was very dyspnœic, but there was less sputum, and the cough was not so troublesome. The activity of the disease was checked and the case developed into one of the chronic fibroid type, the patient being still alive eight years later.

Saugman (7) describes seven cases in which he made a bilateral pneumothorax. One had the left lung collapsed from November, 1912, to July, 1913, and the right lung from January, 1914, to June, 1915. Since then he has been in good health and was known to be still at work in 1919. All the other six cases died.

The contra-indications for artificial pneumothorax are :—

1. In the absence of any of the special indications mentioned, pneumothorax should not be induced if the lesions are healing under ordinary treatment and the patient can afford the time to allow arrest to take place.

2. When there is so much adherent pleura that it is possible to produce only a partial pneumothorax which is not sufficient to rest the diseased part. Sometimes a very partial collapse will have a beneficial effect on the disease, but if it has no effect it is not wise to maintain it. One should remember that a satisfactory collapse is often obtained although physical signs, X-ray or a history of repeated attacks of pleurisy, or even pleural effusion may suggest that there are extensive adhesions. One should never assume that pneumothorax is impossible until one has tried to induce it.

3. In very advanced cases pneumothorax with the necessary refills, often adds to the patient's discomfort, and should not be induced as a last resort when there is no hope for the patient.

4. Patients with a highly neurotic temperament do badly with pneumothorax as with any other treatment. I have had to discontinue the refills owing to the mental state of the patient.

5. It is obviously unwise to induce pneumothorax when the tuberculosis of the lungs is a terminal phase of some intercurrent disease. At the same time, intercurrent disease need not of itself be a contra-indication. For example, many patients with diabetes and tuberculosis do extraordinarily well with insulin and pneumothorax. Asthma and emphysema are not always absolute contra-indications, although in such cases one often has to stop the treatment owing to dyspnœa, but I have known asthmatic consumptives successfully treated by pneumothorax.

6. Active bilateral disease is a contra-indication only if collapse of one lung aggravates the disease in the other and

collapse of both lungs either simultaneously or alternately is not feasible.

APPARATUS

There are many different kinds of apparatus designed to induce artificial pneumothorax, and after a time the physician gets accustomed to a certain type of instrument, some liking one and others another, but really it is a matter of small importance as to which is used. When asked to advise in the choice of a pneumothorax apparatus I always recommend the simplest for hospital and institutional use, and for private work the most portable. The apparatus devised by Drs. Lillingston and Pearson and shown in Fig. 15, is very simple and satisfactory. It consists of two bottles A and B. A is graduated from 0 to 1,100 c.cm., and is tall and narrow, so that the graduations are easily read. Both bottles are fitted with rubber stoppers. A glass tube C passes through one stopper to the bottom of bottle A and a similar glass tube D passes through the other stopper to the bottom of bottle B. These two tubes are connected by a rubber tube E. Shorter glass tubes FF pass just through both stoppers. Now if one bottle and the tubes CDE are filled with water the water will flow into the other until it is on the same level in the two bottles. Air will enter through tube F in the emptying bottle and be forced out through tube F in the other bottle. Tube F from the graduated bottle A is connected by a rubber tube with one limb of a cross-shaped glass tube G. The other three limbs of this glass tube are fitted with rubber tubes, one of which H is connected with the manometer, another J, is connected with the needle and, the third K, is a short tube used only for filling the apparatus with air.

If the tube K is clamped and the bottle B raised so that water flows into A, the air in A will be forced out through the needle at the end of tube J, but if J is also clamped, the air will force down the column of liquid in the limb L of the

manometer and the liquid in the limb M will rise. NO are filters of sterilised cotton wool. P is a short glass tube to act as a window in the tube J near the needle. R is a scale by which the pressure can be read in centimetres of water. STX are clamps. When the apparatus is not in use the open

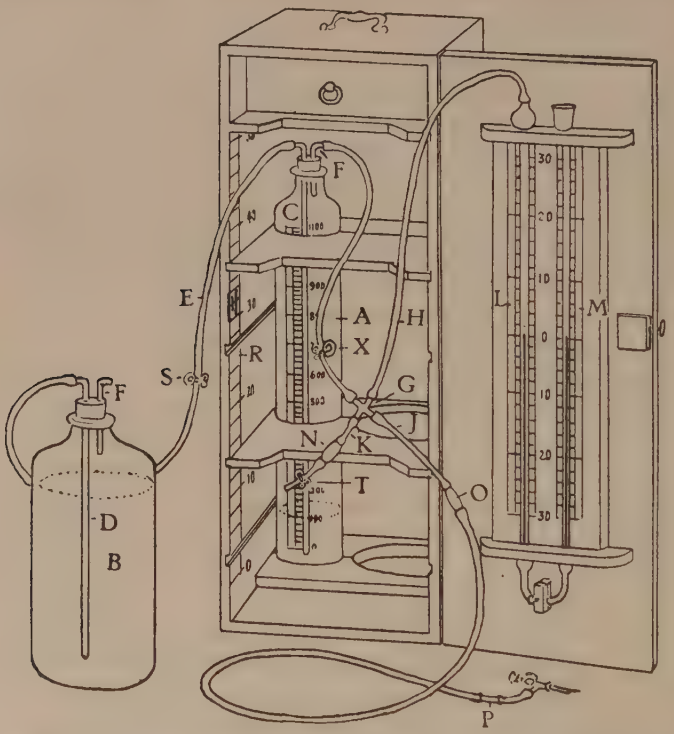


Fig. 15.—Lillington and Pearson's artificial pneumothorax apparatus.

ends of tubes J and K are closed by sterilised glass rods. The apparatus is very simple, the bottles can easily be cleaned and the tubing boiled. The limb of the manometer should be long and fitted with bulbs at the tops so that the liquid may not be forced out of M by a high positive pressure or sucked into tube H by a negative one. I use water

coloured with a little red ink for the manometer liquid. Mercury is too heavy.

Before using the apparatus it is important to see that there is no leak. This can easily be done by making a small negative or positive pressure recorded on the manometer and after clamping the tubes seeing if any air escapes.

In order to use the apparatus, bottle B is filled with 1 in 100 carbolic solution slightly coloured to make it more visible. A little air is blown with a Higginson syringe through F into bottle B to force the liquid through tubes DEC into bottle A, and thus start a syphon action. When the liquid reaches 0 in bottle A, the tubes E and K are clamped. J is clamped at X so that the needle is in connection only with the manometer. The trocar is removed from the needle and the tap at the top turned off. Clamps S and X are now unfastened and the end of the needle is put into a little methylated spirit through which bubbles of air should pass if the apparatus is working properly. Liquid should be allowed to flow from bottle A into bottle B until its level is equal in the two bottles, and when E is unclamped the pressure in the tubing is atmospheric. Thus, air cannot be forced out through the needle under pressure with the risk of causing gas embolism. The tubing is now clamped at S and X (T is already clamped), the needle is dried over a spirit flame and the apparatus is ready for use. For the initial induction of pneumothorax I use a Riviere needle, as this has a blunt end when the trocar is removed, and is less likely to injure the visceral pleura than the pointed Saugman needle, which I use for giving refills. I do not think any other instrument is better than this for hospital use ; besides being effective it is cheap and simple, and any part that happens to get broken can easily be replaced. For private work, however, when the instrument has to be carried from place to place and taken in trains and cars it is rather awkward and heavy, and the liquid is apt to spill. Dr. Heaf (1) designed a portable apparatus

which I have found very satisfactory. It is shown in Fig. 16, and has the advantages of having no liquids in any part and being light and portable. It consists of a collapsible bellows supported by three guides and arranged so as to fall steadily under the weight of a metal cap attached to the tubes which slide over the three guides. The bellows hold 500 c.cm. of air and a graduated scale shows the amount of air used. At the top of the bellows is a metal tube and tap so that they

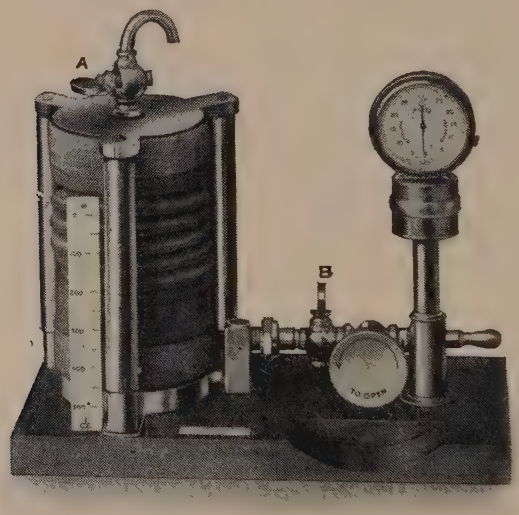


Fig. 16.—Heaf's artificial pneumothorax apparatus.

can be filled with air or other gas, whilst at the base is an outlet which leads the gas through a second tap and reducing valve, and a manometer is fixed at this point in direct connection with the pneumothorax needle. A simple filter is placed between the manometer and the needle. To use the apparatus tap B is closed and the tap A opened. The bellows are raised by the hooked tube until the stop is reached when tap A is closed. The reducing valve is then closed, and the manometer adjusted to zero. When the needle is

inserted into the pleural cavity the pressure can be read on the manometer which is very delicate and, on opening tap B and unscrewing the reducing valves, air will enter the pleural cavity, its rate being regulated by the valve. The apparatus can be taken to pieces quite easily and the inside bellows cleaned with spirit.

Choice of Gas.—I always use air for ordinary pneumothorax work not only because it is easy to get, but because it approaches most nearly to the alveolar air and therefore less interchange of gas takes place in the pleural cavity. Rist and Strohl (2) concluded that there is a balance between the gases of the pneumothorax cavity and the gases in the venous blood. When either air or nitrogen is introduced there follows a short period during which the total gas in the pneumothorax cavity is increased before interchange of gases establishes a balance. Tobiesen (3) found on analysis N_2 90 per cent., CO_2 6·7 per cent., O_2 3·4 per cent. in the pneumothorax cavity whether nitrogen, carbon dioxide or oxygen had been used at the previous refill. Oxygen is absorbed rather more rapidly than air or nitrogen, and is therefore the best gas to use if a rapid absorption is required, as, for example, in certain cases of pleural effusion. It has been used at the initial induction of pneumothorax on the assumption that it is less likely to cause gas embolism owing to its rapid absorption. For a similar reason carbon dioxide, which is readily absorbed by the blood serum, has been used. With proper technique gas embolism should not occur and the choice of gas in this respect makes no real difference. Helium has been tried, but was found to have no advantages and to be absorbed as quickly as air or nitrogen. Nitrogen is still used by some, but I think air is better. Perhaps the American was right when he said that the difference between the two was only a matter of dollars.

The following tables suggest that there is no appreciable difference in the rate of absorption between the two gases :—

1. One of my cases having refills at three-weekly intervals.

March	10th	.	−6	−0	..	700 air	..	+4 + 9
,,	31st	.	−6	−1	..	750 ,,	..	+4 +10
April	21st	.	−5	−0	..	750 N_2	..	+3 + 9
May	12th	.	−6	−0	..	700 air	..	+4 + 9
June	2nd	.	−5	−1	..	700 ,,	..	+4 + 8

2. One of my cases having refills at four-weekly intervals.

April	7th	.	−8	−2	..	1,000 air	..	+2 +12
May	5th	.	−8	−2	..	1,000 ,,	..	+1 +11
June	2nd	.	−7	−3	..	1,000 N_2	..	+2 +11
,,	30th	.	−8	−3	..	1,000 air	..	+1 +11

The rate of absorption varies enormously in different individuals and, as a general rule, it is absorbed more quickly in the early stages of pneumothorax than in the later ones.

TECHNIQUE OF INDUCING ARTIFICIAL PNEUMOTHORAX

The mental attitude of the patient is of the greatest importance. In a hospital or sanatorium where several patients are having pneumothorax treatment the immediate benefits are so obvious to all, and the inconveniences of the refills so slight, that patients often ask to have it and become very disappointed and depressed if told they are unsuitable. Nowadays it is so widely known that the majority of patients expect that it should at least be mentioned as one of the possibilities of treatment. There are still a few, however, who regard pneumothorax as an operation only to be considered as a last resort or as a kind of experiment, and who work themselves up into a state of nerves so that there is a considerable rise of temperature on each day the refill is due, even if for some reason it is not given. A little dyspnœa becomes serious gasping for breath, relieved at once by morphia, a little pain becomes agony and a feeling of tightness in the chest may lead to most alarming symptoms of

distress which are never seen in the patient with confidence. On two occasions I have had tc stop the refills owing to the mental condition of the patient. In such cases it is almost essential for the patient to have a preliminary course of sanatorium treatment and, after a week or so, if he does not himself suggest pneumothorax, he will usually welcome rather than dread the idea of it. Unfortunately this type of patient is the one who so often refuses to go into a sanatorium, and it may be quite impossible to get him into the right frame of mind for any form of real treatment, though a serious attempt should be made to influence him and gain his confidence before starting a pneumothorax.

In any case, the procedure should not be in the nature of a surgical operation and should be done in the patient's room and not in an operating theatre, although of course all instruments should be boiled and the strictest precautions taken against sepsis. It is unnecessary to make any special preparation of the patient or to give him a preliminary dose of morphia or sedative. In case of accidents, brandy, pituitary extract and other stimulants should be in readiness and hot water bottles at hand, but these precautions should be taken without the patient's knowledge and to him the whole procedure should be made to appear no more than an ordinary hypodermic injection.

Site of Puncture.—The site of election is the fifth intercostal space in the mid-axillary line. If the pleura is adherent in this area, so that the lung will not collapse, an attempt should be made in the sixth space in the posterior axillary line and, failing this, in the seventh space, just below and internal to the angle of the scapula. If the pleura is adherent at all these sites, I try in the first intercostal space below the clavicle. Should the pleura be adherent here also, it is not necessary to make any further attempts to induce a pneumothorax, for with such widespread adhesions any collapse obtained would not be sufficient to be of value.

If the skin and pleura are properly anæsthetised with

novocain and sharp needles are used, the punctures are painless, and it is possible to make all four attempts at one sitting. In the case of a patient who shows signs of nervousness it is sometimes best to stop after the second attempt, and to postpone the other punctures for a day or two; it is hardly ever necessary to do this, however, and the great majority of patients prefer to get the whole proceeding over at one time. Only a few patients get a satisfactory collapse after failure to produce pneumothorax at the first attempt, and a successful collapse after a second failure is rare.

There are several advantages of the axillary site for making the first attempt, in the first place the ribs are far apart here, and there is little muscle tissue covering the chest. Secondly, adhesions are less likely to be found here than at other sites, and, thirdly, the lung excursion is greatest here.

In a case where much pleural adhesion is suspected, but where a friction rub is heard, an attempt should be made over the area of friction, for this indicates the absence of adhesions. Whatever the clinical or X-ray findings, one should never assume the presence of too many adhesions to allow a pneumothorax, for one often obtains quite a good collapse in the most unlikely cases. When trying to induce a pneumothorax it is best not to puncture the pleura over a large cavity or over an area of extensive caseation.

The patient should be arranged comfortably in bed, supported by pillows so that the site of the puncture is uppermost and the ribs in that region are as far apart as possible. The area selected for puncture is then cleaned and $\frac{1}{4}$ c.cm. of a 2 per cent. solution of novocain injected intracutaneously so that a small lump is raised and the skin anæsthetised. The hypodermic needle is then inserted into the middle of this lump and pushed slowly down between the ribs, novocain being injected all the time, the last $\frac{1}{2}$ c.cm. against the pleura. This procedure is perfectly painless if the needle is small and sharp. I use a Record needle, size No. 20.

The pneumothorax apparatus, which has previously been got ready and tested as already described, is then arranged at the patient's bedside. If Lillingston and Pearson's apparatus is used (Fig. 15, p. 142) clips SXT are fastened so that when the chest is punctured the pleural cavity will be in connection only with the manometer and not also with reservoir A. For a refill the liquid in A should stand at 0, but for the initial induction of pneumothorax the liquid in A should be at the same level as that in B, so that on releasing clips S and X air can be sucked from A into the pleural cavity by negative pressure, but cannot be forced in by the rising liquid in A.

For refills Saugman's needle, which has a sharp point, is satisfactory, but for the initial injection it is better to use Riviere's needle which consists of a blunt-ended cannula, through which passes a sharp-pointed trocar. In choosing a needle I should avoid one with too small a bore, as the movements of the manometer are not so readily registered with small-bore needles.

For the initial induction of pneumothorax a Riviere needle is fixed to the end of tube J and pushed gently through the anæsthetised area of skin between the ribs and down to the pleura. Provided the needle is sharp, this is quite simple, and it is not necessary to make a preliminary incision of the skin with a tenotome. On reaching the pleura the trocar is withdrawn, the tap at the top of the needle turned off. The blunt end of the cannula is then gently pushed through the parietal pleura, and when it is in the pleural cavity the manometer will show a negative pressure with oscillations corresponding to inspiration and expiration. At this stage the patient must keep absolutely quiet and the needle be held perfectly steady or it is liable to slip out of the pleural cavity. When, from the oscillations, it is certain that the needle is between the visceral and parietal layers of pleura, clips S and X are unfastened and about 50 c.cm. of air allowed to be sucked into the pleural cavity; this allows a

small air space to collect round the needle since the patient has been so arranged that the site of puncture is uppermost. If the manometer still shows a negative pressure with oscillations, it is now safe to raise bottle B and allow some 300 c.cm. of air to enter the pleural cavity. The readings of the manometer are then noted and the needle withdrawn. The exact quantity of air which is introduced will depend on the intrapleural pressure, but for the initial injection 300 c.cm. may be taken as a good average as it is not enough to cause a reaction and yet sufficient to leave an air space which lasts until the first refill on the following day. It is best not to put any dressing over the small puncture which will heal without leaving a scar.

Sometimes when the needle enters the pleural cavity there is registered a negative pressure, but the oscillations soon stop. This is because the visceral layer of pleura tends to obstruct the end of the cannula. At first the small quantity of air in the needle and tubing is sucked into the pleural cavity, producing a bubble, but as this spreads away, the visceral pleura blocks the end of the cannula. It sometimes happens that during inspiration the pressure becomes more negative, but during expiration the visceral pleura blocks the end of the cannula, so that there is a valve action which practically makes the manometer a minimum pressure one.

If the lung is punctured by the needle, air may escape from the alveoli and produce an air space so that oscillations occur as in an ordinary artificial pneumothorax. These small punctures soon heal and, as a rule, do no harm, but they are especially liable to occur if a sharp needle is used for the initial injection.

When the needle enters the lung or a bronchus or cavity there may be oscillations registering a negative pressure during inspiration and a positive one during expiration, but with the mean pressure at zero. If air is introduced it will not affect the pressure in such a case, and I have seen

one where 2,000 c.cm. of air were injected and yet the oscillations of the manometer ranged a point or so above and below zero, because, of course, the air was not entering the pleural cavity, but escaping through the bronchi.

Should the needle pierce the diaphragm and enter the abdominal cavity the oscillations will be inverse; that is to say, a low pressure will be registered with expiration and a higher one with inspiration.

Sometimes the manometer registers a positive pressure which becomes gradually higher. This may be due to the needle being in a blood vessel when blood will pass up the tubing and be seen through the glass tube P. It is in such a case that there is a danger of gas embolism if air is forced in under pressure. I have had cases where the needle has entered a large vessel or the heart, and blood has rushed up the tubing, but no ill effects have followed.

Refills.—It is on careful spacing of the refills and giving the correct quantity of gas that success or failure of pneumothorax treatment depends. When a lung is collapsed no special skill is required to put a needle into the pleural cavity and inject gas, but it requires considerable experience to judge how much to give and when to give it.

A common mistake is to give a definite quantity of gas at fixed intervals regardless of the fact that the right amount varies as treatment proceeds, so that a patient may require more gas at one time and less at another.

Another mistake is to leave too long an interval between the refills so that the lung re-expands sufficiently for the visceral pleura to come in contact with the parietal pleura and to become adherent in places. Firm adhesion may occur in three days or less, and no amount of intra-pleura pressure will separate the adherent surfaces. Even at operation it is often found impossible to do so without tearing the lung, so firm are the adhesions. The process gradually spreads until the whole of the pleura becomes adherent and the pneumothorax cavity obliterated (10) in spite of maintaining high

intrapleural pressures or injecting gomenol into the pleural cavity. Dr. Lillingston (15) describes a case in which the refills were being given once a month, but were left once for an extra two weeks, when it was found that 300 c.cm. of gas caused a pressure of +8. Frequent refills were given and the pressure taken to +16, but the pneumothorax cavity became obliterated. Sometimes one wants the lung to expand in this way and a process of gradual obliteration of the pneumothorax cavity with subsequent fibrosis is often a satisfactory termination of the treatment, but if it is desired to maintain a pneumothorax it is of the utmost importance to prevent the visceral and parietal pleura from coming into contact with each other.

Perhaps the most common mistake is to maintain the wrong degree of collapse. Some patients do best with a very partial collapse, and if this is increased they begin to get thin, develop dyspnœa and lose ground. Others fail to improve until a complete collapse is obtained and large and frequent refills are given. Some of my best cases had regular refills of over 1,000 c.cm. with full collapse of lung and often considerable displacement of the mediastinum. In the majority of cases, however, small refills are more suitable, and I rarely give more than 300 c.cm. for the initial injection. The first refill should be given twenty-four hours later, and subsequent small refills every few days so that the desired collapse is obtained in about a fortnight. The following may be taken as typical examples of the first few refills :—

1.	July 18th	— 6 — 4	300 c.cm.	— 5 — 2
	,, 19th	— 6 — 4	400 ,,	— 5 — 1
	,, 21st	— 6 — 2	500 ,,	— 4 — 1
	,, 24th	— 5 — 2	500 ,,	— 4 — 0
	,, 27th	— 4 — 2	500 ,,	— 2 — 0
	,, 31st	— 2 — 1	500 ,,	— 1 + 1
	August 5th	— 4 — 2	600 ,,	+ 1 + 4
	,, 12th	— 4 — 1	600 ,,	+ 2 + 4
	,, 22nd	— 3 — 0	600 ,,	+ 4 + 7

2.	February 2nd.	−14 −10	300 c.cm.	−10 − 4
	,, 3rd .	−12 − 8	400 ,,	− 8 − 2
	,, 5th .	− 8 − 4	450 ,,	− 6 − 0
	,, 8th .	− 9 − 2	500 ,,	− 4 + 2
	,, 12th .	− 8 − 1	600 ,,	+ 0 + 5
	,, 16th .	− 6 − 0	650 ,,	+ 1 + 6
	,, 23rd .	− 3 − 0	600 ,,	+ 2 + 8
	March 4th .	− 2 + 1	600 ,,	+ 4 + 9

All pressures are centimetres of water.

Yet another mistake is to leave too long an interval between refills in a patient when the gas is quickly absorbed. In such a case there is considerable re-expansion of lung between the refills and instead of keeping the lung at rest (which after all is the main object of the treatment) it is alternately collapsed and expanded. I have spoken of this as the concertina type of case, and, to keep the lung steadily collapsed, small refills should be given sometimes as frequently as every other day. The following is an example of faulty intervals between refills ; the patient had been under treatment by artificial pneumothorax for two and a half years and was having 1,500 c.cm. of air every five weeks. At the end of this time, however, the lung had completely re-expanded. It was found that 600 c.cm. left a pressure of −6, but caused a good collapse which lasted for a fortnight, when the lung began to re-expand quickly. To keep the lung at rest, therefore, 600 c.cm. of air should have been given every two weeks.

When, at the end of about a fortnight, the optimum degree of collapse has been obtained, refills should be regulated according to the rate of absorption of gas and the condition of the patient, and it must be remembered that even in the same individual the intervals between refills and the correct quantity of air required vary as the treatment proceeds. By optimum degree of collapse is meant that which best suits the particular patient, some do best with a very partial and others with a complete collapse. The next thing to discover

is how long this optimum degree of collapse will last without a refill, and this can be done by watching the degree of re-expansion of lung by X-ray, and by observing any return of symptoms.

If, after a refill leaving the intrapleural pressure at —2, the patient is free from symptoms, but with any further collapse he does not do so well and begins to lose weight or have dypnœa, we may regard —2 as the maximum pressure which should be reached for that patient at that stage of the treatment.

In the same way the minimum pressure may be found, a lower one producing a return of symptoms or failure to give sufficient rest to the lung. Suppose —12 is the minimum pressure, then in this case the optimum degree of collapse is that which exists between —2 and —12. The refills and the quantity of gas required can be arranged accordingly. It is well to watch the lung by X-ray screen at frequent intervals in the early stages of treatment to see that there is not sufficient re-expansion to cause danger of adherence of the pleura.

Adhesions may prevent a satisfactory collapse and lead to high intrapleural pressures after only a small quantity of air has been given. If it is decided to go on with the treatment, and try to stretch the adhesions, small frequent refills are better than large ones at longer intervals. High intrapleural pressures in such cases are useless when the adhesions are firm, and dangerous when they may be stretched, but are also liable to rupture. Moreover, a high pressure is very quickly reduced as shown in the following cases :—

	Intrapleural pressure.		Six hours later.
1.	+18 +30	..	+1 +8
2.	+12 +20	..	0 +6
3.	+22 +28	..	+1 +5

If high pressures are obtained it is quite common for a little surgical emphysema to develop around the site of puncture from ecapes of the gas, and this, of course, also lowers the intrapleural pressure.

When fluid develops during pneumothorax treatment the gas is less quickly absorbed, and the weight of the effusion empties the alveoli completely of air, so that the lung is often collapsed for a long period without a refill. It is important, however, to keep these cases under careful observation, for adhesions are liable to form under the fluid and to draw out the base of the lung, thus starting a process of obliteration of the pneumothorax cavity. To prevent this the fluid should be removed or replaced with air if there are any signs of re-expansion at the base of the lung.

In a straightforward case, therefore, it may be said :—

1. That the lung should be allowed to collapse gradually.

2. That when it has been collapsed the optimum pressures should be determined for each individual case and thus correct dosage of gas and intervals between refills ascertained.

3. That the patient must be kept under careful supervision not only because the rate of absorption tends to vary as time passes, but also it may be affected by such complications as spread of adhesions, pleural effusion, etc.

Duration of Treatment.—Professor Saugman (7) wrote : " The pneumothorax being complete I now normally let the patient be treated for about five years. If then all is normal, and if he wishes it, I let the pneumothorax close, choosing the summer season to allow the lung to re-expand, for the first months, observing the patient very closely, if possible in the sanatorium ; and no sooner do symptoms of relapse in the treated lung appear than I renew the injection of gas, but only in a very few cases have I seen reason to do so." He gives the following table :—

Of forty-three patients discharged 1907-1917, and at work in 1919, compression has lasted—

½ year in 1 case.	4 to 5 years in 2 cases.
¾ ,, ,, 2 cases.	5 to 6 ,, ,, 1 case.
1 to 2 years in 11 cases.	6 to 7 ,, ,, ,,
2 to 3 ,, ,, 17 ,,	Unknown in 1 case.
3 to 4 ,, ,, 7 ,,	

Vere Pearson (9) states that in his opinion the compression should be kept up for at least three years if it is to be effective. He would continue treatment with an occasional refill for a further eighteen months if :—

1. The patient is over thirty-three years of age and has lost the resilience and recuperative powers of youth.

2. The compressed lung was a fairly useless one before the initial injection, and if in addition it was producing chronic poisoning.

3. Things are going well during the maintenance of the collapse.

In a large number of cases the termination of the treatment is settled by various factors such as obliteration of the cavity by adherent pleura, development of active disease in the other lung, etc.

If, however, the patient is doing well and keeping free from symptoms and at work I think the collapse should be maintained for three years and then the lung allowed to expand gradually during the summer. Before allowing complete re-expansion I give two or three refills at long intervals, using only sufficient gas to prevent the visceral and parietal layers of pleura from coming into contact, for by this means it is possible to re-collapse the lung fully should a return of symptoms make this advisable.

In cases where there are many adhesions but sufficient collapse to relieve the symptoms, the refills should be kept up whilst the pneumothorax cavity is contracting as long as they are doing good, but when the amount of collapse is so small, that it has no effect on the symptoms, the refills should be stopped.

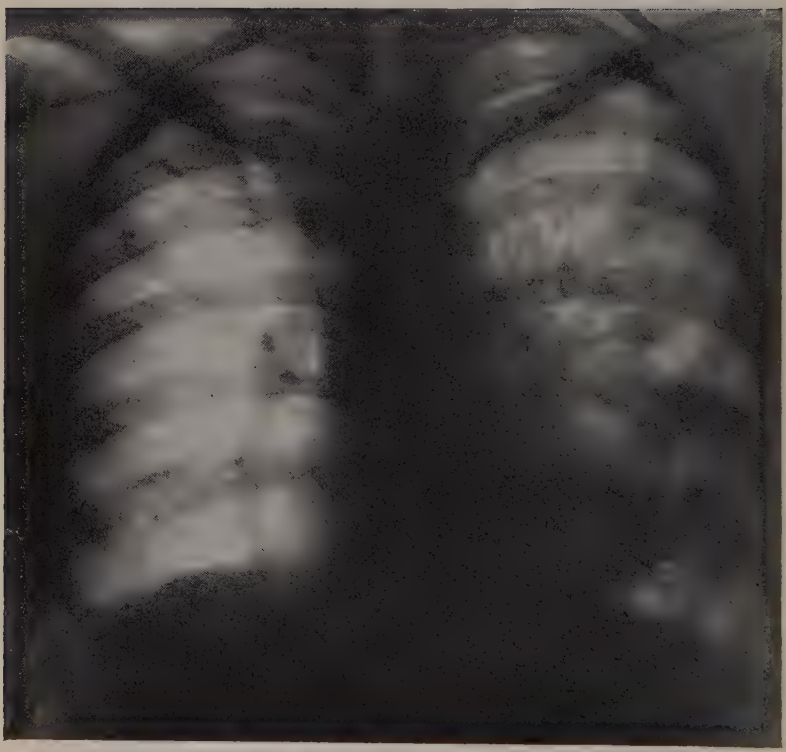
Selective Collapse.—Plate XXV shows a lung with good collapse at the apex except where the lung is slightly drawn out by an adhesion going to the axilla. The base is sufficiently collapsed to keep the lung from the chest wall but is more expanded than the apex. This condition is known as selective collapse, and it has been found that collapse tends

PLATE XXV

Good Apical Collapse, but Partial Expansion of Lower Zone.

[*To face p.* 156.

PLATE XXVI



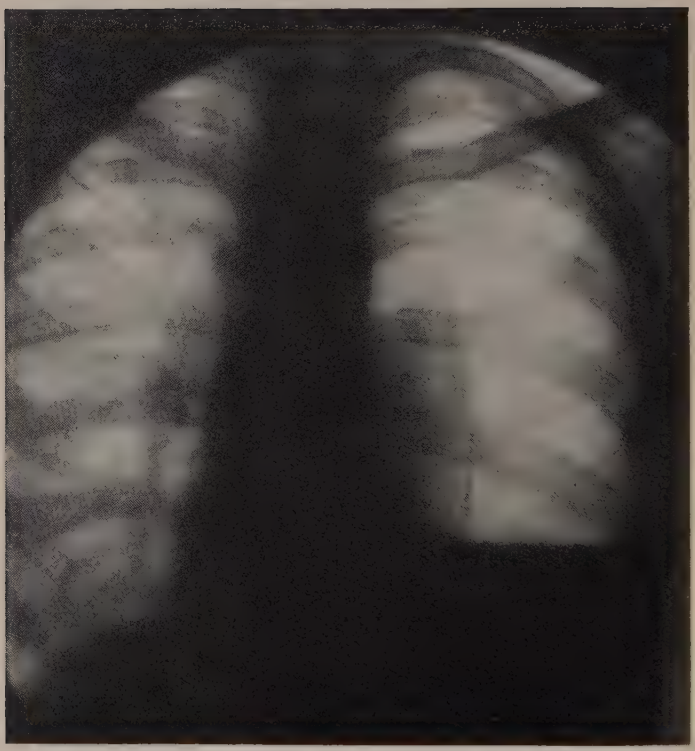
Same as Plate XXV Two Years later. Right lung has re-expanded, but disease has developed in the left lung, chiefly at the base.

PLATE XXVII

Same Case taken Three Months after Plate XXVI and showing that the Collapse is chiefly of the Diseased Area, namely the Base.

[*To face p* 156.

PLATE XXVIII



Same Case. A small effusion has developed, and the lung is now completely collapsed, whereas before the effusion it was only the diseased basal area that collapsed (Plate XXVII), and the diseased apical area (Plate XXV).

to occur in the diseased rather than in the healthy parts of the lung.

Plate XXVI shows the same case twenty-two months later when the right lung has completely expanded but disease has broken out in the left lung and chiefly at the base.

Plate XXVII, taken three months later, shows that on producing a left pneumothorax it is the base that is chiefly collapsed.

Plate XXVIII shows the complete collapse of lung which resulted from the development of an effusion.

It was suggested by Dr. Parry Morgan (11) that a partial pneumothorax would produce the same results as a complete one by resting the diseased part, he pointed out that during inspiration the normal parts of the lung expand against the chest wall, so that a small quantity of air in the pleural cavity would tend to collect over the less expansile or diseased portions. Some hold the opposite view that the normal parts of the lung collapse more readily. In practice the matter is generally complicated by adhesions which are especially to be found over the diseased parts, but there is no doubt that in the absence of adhesions a small quantity of gas introduced into the pleural cavity does tend to collect over the diseased area, and that if a complete pneumothorax is produced the normal portions of lung re-expand more readily than the others when refills are stopped.

In America, Barlow and Kramer (12) have practised selective collapse with good results, and claim that it does not affect the other lung or cause any reaction.

Dr. Riviere (13), after a fair trial of it, has not been able to record any very striking results. He points out that there are several difficulties and disappointments in connection with partial collapse.

Firstly, success depends on a free pleura over the diseased area.

Secondly, even if a selective collapse is obtained, it is very difficult to maintain it at the same amount, and even X-ray

examination may be misleading for the gas so often lies in front of or behind the lung instead of around it.

Thirdly, the treatment is almost always affected by reactions following the refills. He remarks that although in his experience those reactions constitute the main trouble in selective collapse they are not referred to by those who advocate the treatment.

Fourthly, the refills have to be small and frequent and therefore much time has to be expended on each individual case. "Fifteen such cases will take up the entire time of anyone," said Nathan Barlow.

In my experience, a partial or selective collapse may be useful in bilateral cases when it is obtained on both sides at once, but as already explained (p. 138) I usually prefer to collapse the two sides alternately and not simultaneously. Moreover, in bilateral cases it is very rare to find the affected areas free from adhesions on both sides of the chest.

In the acute type of case, which is so common in children about puberty, I had great hopes from selective collapse, but was disappointed for the disease is usually too scattered for a satisfactory collapse of part of the lung to be of value, and as full a collapse as possible should be obtained in such cases. With regard to the ordinary apical unilateral case I think a complete collapse is safer than a partial one, not only because it tends to prevent spread of disease in the healthy part of the lung, but because in partial collapse adhesions commonly form below and around the collapsed part, so that it is impossible subsequently to produce a complete pneumothorax even if it is desired to do so. I quite admit that in certain cases where only a partial collapse was possible owing to adhesions, good results were obtained, but in my series of cases far better results followed full collapse of the lung, and I think this should always be obtained when possible.

Intrapleural Pressure.—Intrapleural pressure is measured by the manometer. Water is generally used as mercury

is too heavy, but some physicians use spirit which gives large oscillations. All pressures given in this book are centimetres of water.

The size of the needle is important, as the oscillations of the manometer are not well shown if too small a needle is used. If two needles (one being a large pneumothorax needle and the other a small hypodermic one) connected with the manometer by means of rubber tubes and a T-shaped glass rod are put into a distended rubber bladder, it will be found that the pressure registered through the small needle is the same as that registered through the large one, but it takes longer to register through the small needle. By giving the bladder little taps, the manometer shows good oscillations if connected through the large needle, but hardly any movement if connected through the small one. Similarly, it will be found that if the liquid in one bottle of the pneumothorax apparatus is higher than the liquid in the other and the outlets are stopped, the column of water in the manometer will show the pressure, and it will be the same whether the tube to the manometer is fully patent or partially obstructed, but it will take longer for the pressure to be registered if the tube is partially obstructed. It is very important that the respiratory oscillations should be well shown, and for this reason the bore of the needle should be at least 1·2 mm.

Normally the intrapleural pressure is negative owing to the elasticity of the lungs and becomes more negative during inspiration. The difference of pressure between full inspiration and full expiration varies in different patients, but it is often considerable and, of course, is largely dependent on the depth of respiration.

Posture has a big effect on intrapleural pressure (14) and this should always be borne in mind when giving a refill to a strange patient and he should be put in the same position for the refill as previously. Some physicians give refills with the patient sitting on a chair, others with him

lying flat on his back, and others with him lying completely or partially on one side. If one has a record of previous refills it is usually possible to tell in what position they were given by putting a needle into the pleural cavity and moving the patient until the usual pressure is found.

If the patient lies on the pneumothorax side, the weight of the viscera will render the pressure in the pneumothorax cavity higher than if he lies on the healthy side. In one of my cases the pressure was -4 $+2$ with the patient lying on the left (pneumothorax) side, but -10 -0 when lying on the healthy side. After a refill of 450 c.cm. the pressures were $+1$ $+3$ and -5 $+2$, respectively. In other words, when this patient was lying on the healthy side he could raise the pressure in the pneumothorax cavity as much by turning on to the treated side as by having a refill of 450 c.cm. It is possible, therefore, to use posture if we wish to stretch adhesions, for by making the patient lie on the healthy side there will be a continual drag on the adherent surfaces. Posture will, of course, have a greater effect if the mediastinum is freely movable and if the lung is not bound to the chest wall by adhesions.

The intrapleural pressure is sometimes lowered by shifting of the mediastinum, and this may occur suddenly. With a very movable mediastinum it is often difficult and sometimes impossible to obtain a good collapse of the lung without displacing the mediastinum and producing symptoms by interfering with the function of the other lung and heart.

Rupture of a pleural adhesion sometimes occurs, and this may affect the intrapleural pressure.

As air is allowed to enter the pleural cavity, the pressure approaches that of the atmosphere, and then if more air is forced in to compress the lung the pressure becomes positive.

The following table shows how the intra-pleural pressure rises as air is introduced :—

	Case 1. *Initial Operation.*	Case 2. *Initial Operation.*	Case 2. *Three Months Later.*
Initial pressure .	−14 −12	−7 −5	− 3 − 0
After 100 c.cm. air .	−10 − 9	−7 −2	− 3 − 0
„ 200 „ „ .	−10 − 8	−7 −2	− 2 + 2
„ 300 „ „ .	− 9 − 6	−6 −2	+ 0 + 2
„ 400 „ „ .	− 8 − 6	−6 −1	+ 1 + 4
„ 500 „ „ .			+ 2 + 5
„ 600 „ „ .			+ 4 + 7
„ 700 „ „ .			+ 6 + 9
„ 800 „ „ .			+ 8 +11
„ 900 „ „ .			+10 +13

	Case 3. *Refill.*	Case 4. *Refill.*
Initial pressure . .	−12 −8	−6 − 0
After 600 c.cm. air .	− 3 −0	—
„ 700 „ „ .	− 2 −0	—
„ 800 „ „ .	+ 2 +2	−2 + 4
„ 900 „ „ .	+ 4 +2	−2 +10
„ 1,000 „ „ .	+ 4 +6	+2 +10
„ 1,100 „ „ .		+4 +10
„ 1,200 „ „ .		+6 +11

In these four cases collapse was not interfered with by adhesions. If adhesions or pleural effusion reduce the size of the pneumothorax cavity, it will sometimes be found that there is a considerable negative pressure at first, but that it rapidly changes. For instance, in one of my cases in which the pneumothorax cavity was gradually becoming obliterated by the spreading of the pleural adhesions, a negative pressure of −24 −12 was found, but it became +20 +26 after 250 c.cm. of air had been introduced.

The following table shows the variation of intrapleural pressure at the initial operation when there are adhesions.

	Case 1.	Case 2.	Case 3.
Initial pressure	−4 − 0	− 4 − 0	−6 −0
After 50 c.cm. air	+8 +12	—	—
„ 100 „ „	—	+19 +23	—
„ 200 „ „	—	—	+2 +6

The next table shows the variation of intra-pleural pressure after the formation of pleural effusion :—

	Case. 1.	Case 2.	Case 3.
Initial pressure	− 6 + 4	− 2 + 4	− 3 − 0
After 100 c.cm. air	− 1 + 6	—	—
„ 200 „ „	+ 8 +14	—	—
„ 300 „ „	+16 +22	—	—
„ 400 „ „	—	+20 +22	—
„ 500 „ „	—	—	+10 +14

When the liquid is replaced by air the quantity removed must be greater than that of the air added if the intra-pleural pressure is to be lowered. For instance, in one of my cases the pressure in the air-space above the liquid was −3 +1 ; after removing 700 c.cm. of liquid it was −14 −6, but on adding 700 c.cm. of air it became +3 +8.

The following cases show the effect of replacing liquid with air on the intra-pleural pressure :—

1. Pressure −2 +4. After removing 1,650 c.cm. of liquid and introducing 1,100 c.cm. of air the pressure was −4 +2.
2. Pressure −8 +3. 1,350 c.cm. of liquid were removed and 1,100 c.cm. air introduced, and the pressure was −8 +2.
3. Pressure −5 −4. 1,250 c.cm. of liquid were removed and 1,000 c.cm. of air introduced, and the pressure was −4 −2.

The lung can remain well collapsed with a negative pressure. Indeed, one almost always finds a negative intra-

pleural pressure when the patient attends for a refill, even if X-ray shows that the lung is well collapsed and that very little re-expansion has taken place since the last refill. A positive pressure left after a refill is soon converted into a negative one. It is not often that one has the chance of seeing how quickly the intrapleural pressure alters, because at the initial operation only a small quantity of air is given and the initial pressure is altered very little, whereas in the later stages of the treatment, when there is a positive pressure, the refills are given at longer intervals. In the two following cases, however, a large initial quantity of air was given to check hæmoptysis, and the pressures were as follows :—

1.	Initial pressure . .	−11	−8
	After 1,400 c.cm. of air .	+ 1	+5
	Thirty-six hours later .	−11	−8
2.	Initial pressure . .	−10	−8
	After 900 c.cm. of air .	− 2	−0
	Two days later . .	− 7	−5

In the following three cases there was much adherent pleura :—

1.	Initial pressure . .	− 9	− 5
	After 120 c.cm. of air .	+10	+14
	Twenty-four hours later .	− 4	+ 1
2.	Initial pressure . .	− 4	− 2
	After 300 c.cm. of air .	+ 1	+ 5
	Sixteen hours later . .	− 2	− 0
3.	Initial pressure . .	− 5	− 2
	After 500 c.cm. of air .	− 0	+ 3
	Twenty-four hours later .	− 5	− 2

It sometimes happens that after a refill the patient complains of dyspnœa and tightness of the chest and a needle is put into the pleural cavity to find the pressure and, if necessary, to remove some of the gas.

1.	Initial pressure before the fourth refill	−3	− 9
	After 700 c.cm. of air . . .	+3	+ 1
	Three hours later	−5	+ 2
2.	Initial pressure before fifth refill .	−5	− 1
	After 700 c.cm. of air . . .	+7	+11
	Four hours later	−0	+ 4
3.	Pressure before initial operation .	−6	− 3
	After 350 c.cm. of air . . .	−4	− 1
	Sixteen hours later . . .	−6	− 4

These cases do not show any evidence of a temporary increase in the volume of gas after a refill.

If a big positive pressure is reached it will sometimes be found that the oscillations recorded by the manometer are reversed—that is, a higher pressure is recorded with inspiration than with expiration. Bjure (25) describes three cases in which the pressure was increased during inspiration, but normal fluctuations were recorded when the patient was told to use costal and not abdominal respiration. The reversed fluctuations in such cases are due to the descent of the diaphragm during inspiration on the healthy side and the ascent on the other side, and this can well be demonstrated by X-rays.

If the pneumothorax needle is accidentally put into the abdominal cavity the readings on the manometer are reversed, the pressure being higher during inspiration.

H. de Carle Woodcock (17), at a post-mortem examination, found an intrapleural pressure of −1·5 in. of water. Intestinal pressure was +2, but after freely puncturing the intestine and allowing the gas to escape, the intrapleural pressure became −3·5. It would appear, therefore, that distension of the bowels had some effect on intrapleural pressure. This may account for the curious changes of pressure very occasionally found at refills. For example, in one of my cases the patient was having 800 c.cm. of air every three weeks, the pressure being taken from about −8 −2 to +6 +10. At the fourteenth refill the pressure

was −2 +4 and 500 c.cm. raised it to +15 +17. No liquid was seen by X-ray, and at subsequent refills the patient was able to take 700 to 900 c.cm. as previously.

It must be remembered, however, that such changes in pressure are almost invariably due either to the presence of an effusion or to extension of adhesions diminishing the pneumothorax space. The development of dry pleurisy may cause a high intrapleural pressure, although no liquid forms. Perforation of the visceral pleura often causes a very high intrapleural pressure. In one of my cases the pressure after perforation was +12 +29, and it was reduced to −6 −1 by removing 1,200 c.cm. of gas.

It has been stated (2) that for a short time after a refill before the gases in the pleural cavity are balanced by interchange with the gases comprising alveolar air, the actual volume of gas in the pleural cavity is increased. This would raise the intrapleural pressure and be one explanation of the dyspnœa not infrequently seen some three or four hours after a refill. It is not my experience, however, to find this rise of pressure.

In some cases, when there was but little collapse before the refill, or more especially at the original induction of pneumothorax, the visceral pleura is pierced and a little air escapes from the lungs; this may continue for some time after the refill and so increase the quantity of gas in the pneumothorax cavity and raise the pressure. I do not think, however, that this is of common occurrence for any slight puncture of the lung usually closes up at once.

It has often been stated that alteration of the intrapleural pressure on one side of the chest affects that on the other, but this is only the case after a certain quantity of gas has been introduced, and it also depends largely on the mobility of the mediastinum. If the intrapleural pressures are taken on both sides of the chest during a refill, it will be found that on the treated side the pressure gets higher and higher as the gas enters the pleural cavity, but that it does

not alter on the other side until a certain amount of gas has entered, and then it will gradually rise as more air is introduced.

In one case of left artificial pneumothorax the intrapleural pressures were as follows :—

Amount of gas.	Left side.	Right side.
0 . . .	−6 −2 ..	−5 −3
600 c.cm. . .	+2 +5 ..	−5 −3
800 ,, . .	+6 +9 ..	−5 −3

In another case of right sided pneumothorax the following intrapleural pressures were obtained during a refill :—

Amount of gas.	Right side.	Left side.
0 . . .	−6 − 4 ..	−8 −6
200 c.cm. . .	+3 + 9 ..	−8 −6
300 ,, . .	+4 +12 ..	−8 −6

Another factor affecting intrapleural pressure is altitude, and this should be remembered in giving refills to those about to go to Switzerland. It is my practice to give no refill to a patient after one full week before he goes to the Alps and then to leave an intrapleural pressure considerably lower than normal. I have known patients soon after a refill arrive at an altitude of some 5,000 ft. with great dyspnœa, necessitating removal of some gas from the pneumothorax cavity. Dr. Riviere (13, p. 103) says that a rise of 3,250 to 5,000 ft. is equivalent in a full pneumothorax of 3 or 4 litres to the addition of 400 to 800 c.cm. of gas. It has been estimated that every 1,000 c.cm. of gas in the chest increase by 40 c.cm. for every 1,000 ft. rise of altitude.

Adhesions.—Collapse therapy is possible only when there is a free pleural space and much the most common cause of failure of the treatment is the presence of too much adherent pleura to allow an efficient pneumothorax to be produced. Cases in which adherent pleura interferes with the treatment may be considered under three headings.

1. When there is too much adherent pleura to prevent any collapse of lung at all or an insufficient collapse to be of any therapeutic value.

2. When there is some adherent pleura, but not enough to prevent a useful degree of collapse. In these cases the value of the pneumothorax is usually impaired to a varying extent, but there is sufficient collapse to give some benefit, and the treatment should be continued.

3. When there are adhesions rather than areas of adherent pleura and although a useful collapse cannot be obtained at first, it is thought possible to stretch or cut the adhesions and so allow the treatment to be given.

It is important to distinguish between adherent pleura and a pleural adhesion, the one may be compared to a ship aground and the other to a ship at anchor. In the one case it is very dangerous to try to separate the adherent surfaces for the lung will almost certainly be torn, in the other it is not difficult to divide the adhesions.

1. When there is too much adherent pleura to allow an efficient collapse.

One should never assume that there are extensive adhesions without trying to collapse the lung. I have succeeded in obtaining a good collapse in cases with much displacement of the heart and trachea, and others with a history of repeated pleurisy or pleural effusion or other conditions which would make the existence of extensive pleural adhesion most probably. There are many signs which make one feel confident that a pneumothorax will be impossible and one is generally right, but it is a mistake to discard the possibility of inducing one without trying.

Unless there are any special reasons for not doing so I make the first attempt in the mid-axillary line in the fifth or sixth intercostal space; failing this I try in the sixth space in the posterior axillary line, then in the seventh space just internal and below the angle of the scapula and finally in the first or second intercostal spaces in the mid-clavicular

line. If no free pleural space is found at any of these sites I abandon the attempt.

In some cases only a small pocket is found and I think it is wrong to keep on refilling it. If the adhesions are firm it is useless to do so, if they are not, it is dangerous, for there is always a chance of it rupturing into the lung, of surgical emphysema, or some other complication. The following cases are examples of such extensive adherent pleura that the pneumothorax treatment should be abandoned.

A. Male aged thirty-six. No free pleural space found at the first attempt. At the second a pocket was found with the pressure −4 −0, but rising to +19 +23 after 150 c.cm. of air had been injected. On the following day a third attempt was made in a different place and failed, but at the fourth attempt a space was found with a pressure −6 −2, but it was raised to +8 +12 by 100 c.cm. of air. No further attempts were made.

B. In this case there was practically no collapse and no benefit to the patient after ten days, so the treatment was stopped. The pressures were as follows :—

August 15th	−6 −3	300	0 + 2
,, 16th	−4 −3	300	+ 6 + 8
,, 17th	0 −1	200	+10 +12
,, 19th	−1 +1	300	+12 +14
,, 22nd	0 +1	300	+16 +20
,, 25th	+1 +3	200	+18 +20

2. When the adherent pleura is not sufficient to prevent a useful degree of collapse. The following may be taken as an example of this type of case.

A woman aged twenty-eight, with slight pyrexia, signs of toxæmia and sputum containing tubercle bacilli. A pneumothorax was induced, but the upper third of the lung was found to be adherent and collapse of the base alone was possible. The temperature, however, subsided, her general condition improved and the quantity of sputum lessened

so the treatment was continued. Gradually, however, the pleural space became smaller and smaller, so that less gas could be given at each refill and the pressures were higher. The treatment was kept up for eight months and had to be stopped then owing to complete obliteration of the pneumothorax cavity. She had, however, gained weight, and lost her toxic symptoms. Four years later she was keeping at work with signs of fibrosis, but apparently no active disease. In this case the pneumothorax was undoubtedly the turning point in the patient's illness, and it illustrates how gradual obliteration of the pleural cavity sometimes gives a good result.

Sometimes, however, as the lung is drawn out, the symptoms return and, in this case, a thoracoplasty should be performed if the condition of the other lung permits. In order to prevent this gradual obliteration of the pneumothorax cavity, frequent refills have been given in the endeavour to keep the intrapleural pressure high. It has already been pointed out that air under pressure in the pleural cavity is quickly absorbed and the pressure falls, so that the gradual obliteration of the pleural cavity proceeds in spite of large frequent refills. It has been suggested that the obliteration may be delayed by injecting liquid into the pleural cavity instead of air. Some liquids, such as normal saline, are absorbed too quickly for this purpose, others set up an inflammatory reaction in the pleura which tends to hasten, rather than slow, the process of obliteration. The liquid most commonly used is a 5 per cent. solution of gomenol in olive oil. This is non-irritant and is slowly absorbed. The quantity given must depend on the size of the pneumothorax cavity and other factors, but it will be found that even when large quantities of oil are injected the cavity will not be filled for the diaphragm becomes depressed and the mediastinum bulges to the other side. It is because of this displacement that oleothorax is not usually satisfactory, and in any case where it is employed the patient must be

watched very carefully lest the formation of an effusion occurs and causes great mediastinal displacement. I saw a patient who had a quantity of air removed from his pneumothorax cavity and replaced by gomenol in order that the lung might remain collapsed during a three weeks' journey to England. On arrival there was extreme dyspnœa and the mediastinum was so much displaced that the heart's beat could be felt in the right axilla. It is important to remember that gomenol should not be given to avoid the necessity of a refill unless the patient is under constant medical supervision.

In some cases the mediastinum cannot be displaced as it is fixed by the adherent pleura and, if this is so, gomenol may safely be left in the pneumothorax cavity and it does undoubtedly delay the process of obliteration. In my experience, however, I have very rarely seen any advantage in maintaining a small pneumothorax. If the symptoms do not return as the lung is drawn out, a good result with fibrosis may be anticipated. If, on the other hand, the symptoms do return, it seems to me safer to advise thoracoplasty, unless the condition of the other lung is too bad, in which case the prognosis is grave whatever is done. In cases where the mediastinum is freely movable, I think oleothorax is contra-indicated.

To introduce the oil two needles should be used, so that air can leave through the upper one as the gomenol enters through the lower. One limb of the upper needle is connected with a manometer so that the intrapleural pressure may be taken from time to time. If the mediastinum becomes much displaced, it will be found that the pressure rises very little as the gomenol is introduced. The same thing is seen when performing gas replacement, for after a certain quantity of liquid has been removed a much smaller quantity of air is required to restore the pressure. A Dieulefoy's aspirator is quite satisfactory for introducing the gomenol solution, which should be warmed and injected

slowly. If there is an effusion it should be completely aspirated before the gomenol is introduced.

3. When the lung is prevented from collapsing by adhesions and not by adherent pleura. If sufficient collapse to relieve the symptoms can be obtained it is not advisable to divide the adhesions. They will tend to stretch under the treatment if small frequent refills are given. There are dangers in giving large refills and making a high intrapleural pressure, and it is not necessary to do so. Even if the collapse seems sufficient the adhesions should be stretched, because results in a case of partial collapse are not so good as those when the lung is fully collapsed; moreover, they may lead to obliteration of the pneumothorax cavity.

If the adhesions prevent an efficient collapse or hold up some part (such as a cavity) which it is desired to collapse, it will often be found possible to stretch them by giving small frequent refills, and when the patient is at rest he should lie on the healthy side as much as possible, as this will produce a drag on the adhesions. In my opinion these methods of stretching them should be given a good trial before resorting to surgical means. Open operation and division of the adhesions is dangerous, and I have seen two patients who died shortly after such an operation, one from hæmorrhage and the other from shock. If the adhesions are to be divided it is best to do so with the cautery under direct vision through the thoracoscope. This method was described by Jacobæus in 1913 (16). After the exact position of the adhesions has been determined, and a refill has been given to raise the intrapleural pressure, a special trocar and cannula are inserted into the pneumothorax cavity. The cannula is made with a valve so that on withdrawing the trocar, no air can escape from the pleural cavity. After withdrawing the trocar a thoracoscope is introduced through the cannula, and a view of the adhesions can then be obtained. When the adhesions are in the upper part of the chest I insert the thoracoscope high up in the back be-

tween the vertebral column and the upper part of the scapula, but the site selected will, of course, depend on the position of the adhesions. On examination through the thoracoscope the collapsed border of the lung will be seen, and leading from it adhesions pass outwards to the chest wall. Some of these are string-like and can easily be divided with the cautery, others are thick with a broad attachment to the chest wall, contain lung tissue, and cannot be divided, others again appear to have lung tissue in them as they leave the border of the lung, but not at their attachment to the chest. This last type is the one which most frequently holds up a large portion of the lung and is, therefore, the one in which it is especially important to beware of cutting through lung tissue. If there is any doubt as to whether or not there is lung tissue in an adhesion, no attempt should be made to divide it. It is far safer to abandon the pneumothorax and advise thoracoplasty or some other treatment.

If, however, it is decided to cauterise some adhesions, another cannula should be inserted through the anterior axillary line (or some other site according to the position of the adhesions) and through this a cautery is introduced into the pneumothorax cavity. With practice it is not difficult to divide the adhesions with the cautery by this method, but it is very difficult to determine whether or not it is safe to cauterise any given adhesion.

Jacobæus (16) describes the method and its results. The most serious complication is empyema, which usually results from injury to the lung and opening up a caseous focus. Lung tissue may be found in an adhesion the size of a pencil. Hæmorrhage into the pneumothorax cavity is another complication, but this can largely be prevented by using the diathermic cautery to divide the adhesions. Bleeding may be considerable and, in one case, was as much as 200 c.cm. Provided proper local anæsthesia is used the proceeding is painless, and it is safe if only the small string-

like adhesions are divided, but this is not enough to free the lung in most cases and it is when the larger adhesions are tackled that the dangers arise. In practice I have found that cauterisation is very seldom indicated, and in most cases, where there are large adhesions which will not stretch, I should prefer thoracoplasty. There are, however, a few cases where very good clinical results follow the division of the adhesions.

Dr. B. Hudson (19) reports a case where improvement followed cauterisation and is of special interest because the adhesions, which were not only thick but vascular, were successfully divided by the diathermic cautery without any bleeding. Dr. Hudson says that in this case the adhesions not only prevented a successful collapse of the upper part of the lung, but were a positive danger owing to the presence of a widely open thin walled cavity with which they were connected.

Dangers of Inducing Artificial Pneumothorax.—Dr. Woodcock (17) wrote in 1915 : "There are dangers in connection with the production of artificial pneumothorax, but the greatest—and about this let there be no mistake—is the neglect in which it is held."

To-day collapse therapy is far more widely employed, and its value is increasingly recognised, but still there are in England many towns, and some of considerable size, where nothing is known about it. I have known a patient who had to travel over sixty miles to get a refill. A few years ago it was necessary to find where the patient lived and whether the refills could be continued at home before starting to collapse the lung, and even now it is sometimes difficult to arrange for a patient's refills after he has left the hospital or sanatorium.

The actual dangers of inducing a pneumothorax under proper precautions are negligible, but unfortunately two of them, namely, gas embolism and pleural shock, though both extremely rare, are very serious and often fatal.

Gas Embolism.—Air may enter the pulmonary veins and, if some bubbles are carried to the coronary vessels causing embolism of the capillaries, sudden death may occur. Hemiplegia or other forms of paralysis may result from gas embolism of the cerebral vessels.

In one case the patient became semi-conscious for a few moments and afterwards for several hours had partial hemianopia and hemiparesis.

In another case, during induction of pneumothorax, the needle was felt to pass the pleura, but no manometer movements were recorded. Oxygen was introduced and the patient at once complained of severe præcordial pain and numbness of one arm. Convulsive movements and unconsciousness followed and the heart and respiration stopped, Recovery soon occurred after artificial respiration and stimulants had been applied, but there was facial paralysis for a few hours. The patient was well the next day.

In another case the patient, a woman of thirty-two, had been under treatment for nearly two years, and the pneumothorax cavity was practically obliterated. The needle was pushed into the lung, for she afterwards coughed up a little blood and the oscillations of the manometer just above and below zero were characteristic of the swing when the needle is in a cavity or lung tissue. At the time, however, it was thought that the needle was in the pneumothorax cavity and some air was introduced under pressure from the apparatus. After about 200 c.cm. had been given she suddenly became faint, complained of pins and needles in the legs and inability to breathe. She then actually lost consciousness for a few moments, but was quickly restored by stimulants. On recovery she complained of weakness and a feeling of numbness in the right leg, but she had quite recovered by the next morning.

It is often difficult to distinguish this condition from pleural shock, and it has been suggested that the great majority of cases described as shock are, in reality, due to

embolism, but with modern methods, gas embolism is very unlikely. Before the manometer was used in pneumothorax work, the needle was gently pushed into the chest until gas flowed from the apparatus, when it was assumed that the needle was in the pleural cavity. It was occasionally in lung tissue or a blood vessel, however, and if the gas entered the pulmonary veins embolism was liable to occur. By using a manometer and not allowing any gas to flow under pressure until it is certain that the needle is in the pleural cavity, gas embolism can usually be prevented, and, indeed, it has been truly said to belong to the historical period of pneumothorax therapy.

Stivelman (18) found no case of gas embolism amongst 867 cases treated by nineteen American doctors.

It has been suggested that air may enter the pulmonary veins from the alveoli when they are punctured. In cases of chronic pulmonary tuberculosis there is free anastomosis between the bronchial (systemic) and pulmonary veins, so that gas entering any intrathoracic vessels may find its way to the systemic capillaries. This is not likely, however, or must be extremely rare as most gas which got into the systemic veins would pass through the right side of the heart or be blocked by the pulmonary capillaries. It is, therefore, only the pulmonary veins that are important in the matter of embolism. These contain arterial blood, so that it is not necessary to use oxygen when inducing a pneumothorax on the belief that it will be more readily absorbed by the blood and so less likely to produce gas embolism. The use of carbon dioxide has also been advocated at the initial induction of pneumothorax because it is quickly absorbed by the plasma, but it is doubtful whether this makes any practical difference and accident should be prevented by correct technique. If the needle does enter a big vessel blood is forced up the tubing and can be seen at the glass window. Any blood entering the needle quickly clots and blocks the needle. When the pleura is adherent the needle

is often pushed, and sometimes deeply, into lung tissue, but no gas can enter the circulation unless it is under pressure. In order to prevent gas embolism, the following precautions should be taken :—

1. Allow the gas to be sucked in very cautiously and use no pressure until the movements of the manometer make it certain that the needle is in the pleural cavity. For this purpose, if Lillington and Pearson's apparatus is used, the level of water in the two bottles should be equal when starting an initial refill.

2. The needle and tubing should at first be connected only with the manometer, being clipped off from the gas chamber.

3. Use a large and blunt tipped needle for the initial induction of pneumothorax.

4. Coughing or deep breathing should be prevented, at least until the needle is in the pleural cavity and the gas flowing freely.

Pleural Shock.—This rare accident may occur when the pleura is punctured or irritated or even when the pleural cavity is being washed out. I have known a case where the patient suddenly died when the chest was punctured for the purpose of aspirating a pleural effusion, and another where sudden death occurred whilst B.I.P. was being introduced into an empyema sinus. With animals it has been found that shock can be produced by irritation of the pleura and probably occurs through the vagus. It can be prevented by ligaturing the carotids, or by keeping the animal under an anæsthetic. Some have denied the existence of pleura shock, claiming that the symptoms are due to embolism, and there is no doubt that the symptoms are very similar in the two conditions. But apart from all other reasons, the fact that shock is liable to occur in the same individual, whenever the pleura is punctured, must exclude gas embolism as the cause of all cases.

The symptoms vary from a mere faintness to actual death and usually occur as the needle is entering or leaving the

pleural cavity, but they may come on during a refill or even some minutes after it is finished. It is said to be more common in the early stages of disease, when the pleura is healthy, than in cases of advanced tuberculosis, and this is one reason why Dr. Riviere (13, p. 19) does not advocate artificial pneumothorax in the treatment of early pulmonary tuberculosis. Pleural shock is rare, and it is not easy to determine whether or not it is less unlikely to occur in cases where there is healthy pleura, but it certainly may occur when the pleura is considerably involved, even in cases of chronic empyema sinus. The following cases are examples of pleural shock :—

1. Stivelman (18) describes the case of a patient with pulmonary tuberculosis who had a fibroma removed from his arm under cocaine and adrenalin on February 1st, and there was no ill effect. On February 20th an attempt was made to induce an artificial pneumothorax and after giving an injection of morphia, $\frac{1}{8}$ gr., the pleura was anæsthetised with 1·5 c.cm. of a 0·75 per cent. solution of cocaine and adrenalin 1 in 8,000, but before the hypodermic needle was withdrawn the patient collapsed, the pulse was only just perceptible, there was stertorous breathing and the patient became livid. Stimulants were given and he recovered after an hour and a half. Two days later he had a similar, but worse attack, whilst the pleura was being anæsthetised, and sent completely blind for four hours. Photophobia lasted twenty-four hours, but he was quite well in two days' time.

2. In one of my cases (4, p. 32) the patient, a man of thirty-eight, was very ill with tuberculosis which had followed pleurisy. The whole of the left lung was involved. He was given an injection of morphia, $\frac{1}{4}$ gr. half an hour before the operation. The track of the needle and the pleura were anæsthetised with a 2 per cent. solution of novocain, and an attempt was made to induce a pneumothorax from the left axilla, but it failed. A second attempt was

made at the back after anæsthetising the pleura as before, but again the pleural cavity could not be found. I was just about to withdraw the needle when the patient suddenly said he could not breathe. There was no movement of the manometer as would have occurred had air been sucked into the blood stream, for the tube leading to the air chamber of the apparatus was clipped and the only air which could have been sucked in was that contained in the tube between the needle and the manometer. I withdrew the needle at once and the patient sat up, gave a few gasps and died. There was no post-mortem examination.

3. In another of my cases, a woman of twenty-eight, pleural shock occurred, but not the first time the pleura was punctured, and, indeed, in this case the pleura seemed to become more sensitive as treatment progressed. During the third refill she complained of a sudden feeling of giddiness. After the needle had been removed following the fourth refill she had a sudden pain over the heart, could not breathe and lost consciousness for about two minutes. During the fifth refill, when about 150 c.cm. of air had been given, she suddenly gasped, became blue and almost pulseless. The needle was withdrawn and stimulants administered and she recovered without having actually lost consciousness. After this treatment was discontinued.

It is doubtful whether anything practical can be done to prevent pleural shock. It is said not to occur in animals under a general anæsthetic, but the danger of complications from an anæsthetic is obviously greater than the risk of pleural shock. A preliminary injection of morphia had been given in the only fatal case in my series and also in several of the other published cases. I always use novocain to anæsthetise the pleura as well as the skin, so that the puncture is absolutely painless, but in spite of this, shock has occasionally occurred in some of my cases, and Stivelman (18) found pleural shock equally frequent whether a local anæsthetic had been used or not in a series of 867 patients treated

by nineteen American doctors. A series of cases treated with certain technique and without pleural shock does not indicate that shock can be prevented by following that method, for it is extremely rare whatever technique is used.

Puncture of Lung.—If the visceral and parietal pleura are adherent it often happens that the needle passes straight through into the lung. In this case a negative pressure may be registered on the manometer, but the oscillations are just above and below zero, so that the mean pressure is 0. The needle usually becomes blocked with clotted blood. A little blood-stained sputum may follow this accident, but there are no other ill effects.

If the pleura is not adherent, it is usually possible to avoid piercing the visceral pleura by being very gentle and using a blunt Riviere needle for the initial operation. In some cases when the lung is punctured air may escape and lead to an increased pneumothorax cavity, and possibly a higher intrathoracic pressure and dyspnœa some hours after the needle has been withdrawn. Usually, however, a small puncture quickly closes and no air escapes from the lung.

Punctures of a Large Vessel or of the Heart.—Vere Pearson (9) describes a case in which the needle entered the pericardium. The manometer showed negative oscillations synchronous with the heart. There were no ill effects.

Minor (8) was inducing a pneumothorax in a young woman with an old fibroid lesion. Suddenly she gasped and turned pale and blood rushed up the tubing as far as the gas chamber. The needle was at once withdrawn and there were no ill effects.

In one of my patients pneumothorax treatment had been stopped two years previously, but symptoms had returned and I was trying to re-collapse the lung. Suddenly the manometer registered a positive pressure which gradually increased and blood began to flow up the tubing and was visible at the glass window beyond the needle.

In two other cases a similar occurrence took place, but

in none of these was there any pain or discomfort at the time, or any subsequent ill effect.

Surgical Emphysema.—Superficial emphysema is not uncommon, especially if there is a high intrapleural pressure, or if the patient coughs during the refill. It is, however, quite a painless and harmless complication and quickly clears up. Deep emphysema is caused by the air entering a false passage between the pleural layers. It travels to the neck along the trachea and œsophagus, and is accompanied by pain and severe dyspnœa. There may also be difficulty in swallowing and a sensation of choking. In three cases in my series the emphysema was severe, but eventually subsided without leaving any harmful after effect. In one patient two attempts to induce an artificial pneumothorax had failed owing to adherent pleura, and twenty-four hours after the attempt the patient complained of pain across the chest, and some surgical emphysema was found in the chest and neck. Two days later it had spread all over the chest and abdomen. Both arms and hands became affected and emphysema could be felt at the very tips of the fingers. The neck, face and scalp became enormously swollen, and he could hardly see out of his eyes. The whole body was distended with surgical emphysema, from the top of the head to Poupart's ligaments, but below this there was no emphysema at all. The patient looked most uncomfortable, and his appearance was alarming, but except for a slight choking feeling he complained of no discomfort. There was no pain or pyrexia, and the emphysema, which began to subside after the third day, had completely disappeared in a week.

Pain.—The treatment itself should be quite painless if the skin and parietal pleura are properly anæsthetised. Even when inserting the large cannula for a thoracoscope there should be no pain. Patients are usually apprehensive at first, but on finding that they feel nothing, do not dread the subsequent refills. Some patients are much more sensitive

than others, and in two cases I have had to stop the treatment as the patient could not endure the refills. One, a man of forty, described the procedure as agony, he clenched his fists during a refill and beads of perspiration came on his forehead although he was always given a preliminary injection of morphia. Of course, if the local anæsthetic is not given properly or if the needle is inserted diagonally so that it pierces a portion of the pleura which has not been anæsthetised there will be pain, but some few patients complain of pain and undoubtedly feel it at every refill to which they never become accustomed. In many cases there is a large nervous element; in one case I had removed the needle after a refill and was holding a pad of wool over the site of puncture as there was a little bleeding. The patient said the pain was so severe he could not bear it any more, and could I stop the refill. I said, "Certainly, I have just finished," and, pinching the skin, removed the pad as if taking out the needle. I then held another pad over the chest, but told him it was only wool, and that the needle was out. He said the pain had gone. In this case he could not feel the needle being withdrawn, he did not even know whether or not it was in the chest, yet he had a feeling of pain amounting to what he called agony as long as he thought it was in the chest. Whether genuine or imaginary, pain does sometimes occur to such an extent that the treatment cannot be continued, though in most cases if a refill is given gently and quickly and the track of the needle is thoroughly anæsthetised there is no pain at all.

Pain after a refill may occur, and it varies from a mere feeling of pressure to an acute pain. A really acute pain is usually due to rupture or tearing of some pleural adhesion, and a sharp pain on cough may be due to stretching of adhesions. A feeling of pressure or discomfort is often due to an excess of air in the pneumothorax cavity, and is generally the result of giving too big a refill. Sometimes, however, extra air gets into the pneumothorax cavity from

the lung which has been accidentally punctured during the refill, or according to Rist and Strohl (2) there may be a temporary increase of gas in the pleural cavity before the balance is established between the alveolar air and the gas introduced.

One may recognise three types of pain following artificial pneumothorax refills :—

1. The patient first complains of tightness in the chest but not actual pain which lasts for a few hours after a refill. It occurs only in patients with adhesions and a high intrapleural pressure, and it wears off as the pressure falls.

2. A pain which comes on some hours after the refill and is increased by cough or deep inspiration. This is often referred to the shoulders and usually to where X-ray shows the presence of adhesions.

3. The sudden snapping of an adhesion. This may occur for no apparent reason, or with cough or during a refill. In one of my cases the patient was a man who had been under treatment for eight months. He was doing very well, was not at all nervous and had never complained of pain before, but during one refill, after about 500 c.cm. of air had been given he suddenly complained of a sharp pain in the chest as if something had torn. It lasted a few seconds only and I was able to finish the refill. It had no effect on the intrapleural pressure. The patient compared the pain to that felt when the dentist touches a nerve. It did not recur at any subsequent refill and was possibly due to the rupture of some small adhesion.

In several cases in my series the patients felt something give inside them, but there was no actual pain. Indeed, in one of these cases the patient had a feeling of tightness and pain which was relieved at once after the sensation of something giving way in her chest.

A sudden pain accompanied by dyspnœa may result from ruptured lung, that is the occurrence of spontaneous pneumothorax in an artificial one. Dyspnœa is usually

the chief symptom, and there may be no pain, but in some cases the pain is so severe that it simulates a perforated gastric ulcer.

Pain occurring before a refill is usually due to pleurisy, the expansion of the lung allowing the visceral and parietal pleura to come into contact. It should be taken as a warning and refills given at shorter intervals or adherent pleura will probably result.

Lastly, pain may rarely be due to deep emphysema and is then felt under the sternum and in the throat. It comes on a few hours after the refill and is accompanied by a sensation of choking and severe dyspnœa. It is very unusual, however, for actual pain to occur in cases of surgical emphysema.

Dyspnœa.—Shortness of breath occurring during pneumothorax treatment may be due to over-collapse of the lung. In cases where one lung is much diseased or collapsed, a pneumothorax on the other side may lead to severe dyspnœa. It is surprising, however, how much collapse in both lungs may occur with little or no dyspnœa, though it is obvious that the degree of collapse which can be obtained in one lung must depend on the amount of functioning lung on the other side. If a large pneumothorax is produced so that the mediastinum is much displaced there may be great shortness of breath, even if the other lung is healthy.

Dyspnœa may also occur when the intrapleural pressure is too low. It may be very severe after or during aspiration of pleural effusion or when air is removed from the pneumothorax cavity. Sometimes when the lung is re-expanding dyspnœa occurs and is relieved by giving a refill.

Sometimes dyspnœa occurs about three hours after a refill, and it has been suggested that this is due to an increase in the size of the pneumothorax owing to interchange between the alveolar air and the gas in the pleural cavity. It is doubtful, however, whether there really is any temporary increase in the quantity of gas, and if it does occur on some

occasions it is more probably due to accidental pricking of the visceral pleura and escape of alveolar air. This type of dyspnœa is largely nervous in most cases, it starts as a little shortness of breath which alarms the patient and makes him more conscious of it until sometimes he is worked up into a state when he is gasping for breath. Reassurance or any medicine given for its mental effect will usually relieve the symptoms at once, though occasionally it is necessary to give an injection of morphia.

Displacement of the Mediastinum.—In most cases of pneumothorax the mediastinum is slightly bow-shaped, the concavity being towards the pneumothorax. Sometimes, however, it is resistant and firmly held in position, so that there is no displacement, even with high intrapleural pressures and complete collapse of the lung. In other cases it is extremely mobile and is displaced even when the intrapleural pressure is negative. In such a case it may be necessary to discontinue the treatment if it is impossible to get a sufficient collapse of lung.

In most cases the optimum degree of collapse can be reached without undue displacement of the mediastinum, but even when there is some displacement I ignore it unless symptoms are produced. I have frequently seen patients steadily improve when the lung is fully collapsed, in spite of considerable bulging of the mediastinum. It is not necessary to interrupt the treatment simply because of a weak mediastinum. Generally, however, symptoms occur and the pressures have to be reduced. A common symptom is loss of weight as illustrated in the following two cases :—

1. A girl of seventeen was admitted to hospital with infiltration of the upper third of the left lung. Before artificial pneumothorax was induced, even whilst she was being treated with absolute rest in bed, the evening temperature was 99·6° to 100·5° F. A left artificial pneumothorax was induced on July 18th, 1921, and the following table shows the changes in the patient's weight :—

	st.	lb.		st.	lb.
July 12th, 1921 .	8	8	Nov. 28th, 1921 .	7	12¾
Aug. 9th ,, .	8	9¼	Jan. 2nd 1922 .	7	9
,, 24th ,, .	8	8¼	,, 24th ,, .	7	9
Sept. 11th ,, .	8	4½	,, 31st ,, .	7	10½
,, 20th ,, .	8	3	Feb. 14th ,, .	7	12½
Oct. 20th ,, .	8	0	,, 20th ,, .	7	12
Nov. 7th ,, .	7	12½	June 6th ,, .	8	5½
,, 14th ,, .	7	12¾			

On October 20th X-ray showed good collapse of the left lung with slight displacement of the mediastinum to the right. The patient was up for six hours a day and had a normal pulse and temperature. The weight continued to drop, however, and in November the patient was put back to bed. This stayed the loss of weight for a time, but in January it began to fall again. At this time X-ray showed considerable displacement of the heart and mediastinum to the right, with bulging of the upper part of the mediastinum. No refill was given from December 30th until February 10th, when the mediastinum had come back towards its normal position. Refills were then given very carefully to prevent displacement of the mediastinum, and the patient began to gain weight.

2. Female aged twenty-one, with infiltration of the left apex. A right-sided pneumothorax was induced on October 27th, 1921, and the patient's weight varied as follows :—

	st.	lb.		st.	lb.
Oct. 22nd, 1921 .	9	6¼	Feb. 14th, 1922 .	8	5
Nov. 14th ,, .	9	7½	,, 27th ,, .	8	5
Dec. 12th ,, .	9	5½	March 13th ,, .	8	5
,, 30th ,, .	9	1½	,, 27th ,, .	8	7
Jan. 9th, 1922 .	9	1	April 24th ,, .	8	7½
,, 24th ,, .	8	12	May 8th ,, .	8	8
,, 31st ,, .	8	8	,, 22nd ,, .	8	8¾

There was the initial gain in weight which usually follows

hospital feeding and rest, but this gave way to a steady loss. On January 30th the mediastinum was much displaced and bulging to the left in the upper part, forming a pleura hernia. By lengthening the intervals between the refills and regulating the quantity of gas given (at the same time taking care that the lung did not re-expand and produce adherence of the two layers of pleura) the mediastinum returned to the normal position and the loss in weight at once stopped.

Dr. Riviere (13, p. 155) points out that although with left-sided pneumothorax a considerable degree of mediastinal displacement may occur with very few symptoms, and often the intrapleural pressure remains low and may even be negative, this is not the case with right-sided pneumothorax. Here severe dyspnœa is common. He suggested that the mediastinum should never be displaced beyond carrying the deep cardiac dulness to the right nipple line in a left-sided pneumothorax, or to the left anterior axillary line in a right-sided pneumothorax, however low the intrapleural pressure may be.

Pleural Hernia.—Occasionally the pleura forms a hernia which protrudes through the mediastinum to the other side, or between the ribs forming a superficial tumour.

The common sites for bulging beyond the mediastinum are in the anterior mediastinum between the second and fourth sterno-costal articulations and in the lower part of the posterior mediastinum. These herniæ can be diagnosed only by X-ray, and cause no symptoms as a rule. In one case I saw a pleural hernia in the fourth left intercostal space in the mid-axillary line. When the patient coughed there was a swelling about the size of a hen's egg in this situation. It could easily be reduced and the air could be heard and felt re-entering the pleural cavity.

Febrile Reaction.—Formerly, when more gas was given at the initial induction of pneumothorax, reactions were common, but now it is rare to obtain even a mild one. At

the same time, symptoms of toxæmia often disappear very quickly after a severe reaction, whereas, now that the lung is collapsed more gradually by small frequent refills, it takes longer for the temperature to fall and the symptoms to subside. The effect of reaction is discussed in Chapter VIII. under Shock Therapy.

Most authors think that reactions should be avoided, and prefer the gradual collapse of lung, and I agree that this is generally the best procedure. It is a mistake, however, to be bound by any hard and fast rule, and if the patient fails to improve with small refills, large ones should be tried and

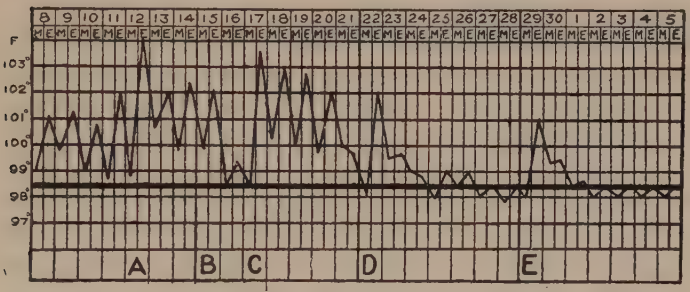


Fig. 17.—A. Initial operation, 400 c cm.; B. 1st refill, 550 c.cm.; C. 2nd refill, 600 c.cm.; D. 3rd refill, 600 c.cm.; E. 4th refill, 700 c.cm.

will often do good in spite of reactions. Fig. 17 shows a series of reactions after refills of moderate amount, and it will be noticed that the temperature soon became level (4, p. 45). The patient now (January, 1929) is well and at work.

Infection of the Track of the Needle.—Ordinary pyogenic infection does not occur if aseptic precautions are taken, but a fistula may follow aspiration of a tuberculous empyema. Sometimes this will heal quickly with a little ultra-violet ray treatment, but often it persists and adds considerably to the gravity of the prognosis.

Perforation of the Visceral Pleura.—It sometimes happens

that a rupture of the visceral pleura occurs and converts the artificial pneumothorax into a spontaneous one. This is a very serious complication, and usually leads to a fatal pyopneumothorax. The symptoms are sudden pain in the chest accompanied by a rise in temperature which becomes hectic. The patient is acutely ill, an effusion soon forms and becomes purulent. The intrapleural pressure may be much increased if the perforation is valvular, and the patient will be very dyspnœic until some gas is removed. Breath sounds over the pneumothorax cavity are amphoric, metallic tinkling is heard, and there is usually much displacement of the mediastinum. Perforation of the lung which occasionally occurs during a refill very rarely leads to any serious consequences, for the small puncture usually closes up at once. The serious rupture is most common where an adhesion joins the visceral pleura and a tear results from the sudden increase of intrapleural pressure during cough. Occasionally a thin-walled cavity or an area of caseating lung may rupture, and it is not uncommon for a pyopneumothorax to rupture through the visceral pleura and leave a gaping communication between the lung and pleural cavity.

Dr. Hutchinson (22) describes a series of cases which he believes to be due to a slight rupture of lung producing an effusion, but soon healing. He points out that an effusion is often preceded by a sudden rise of temperature which gradually falls and is very similar in type to that seen in ordinary spontaneous pneumothorax. Moreover, if the intrapleural pressure is taken in these cases, it is often found to be raised before any effusion has formed. This is a plausible explanation of the rise of intrapleural pressure, which undoubtedly does frequently precede the development of effusion in pneumothorax cases and there is no reason why a rupture should not occur and heal quickly, just as it may do in ordinary cases not treated by artificial pneumothorax. It is very different, however, from the

sudden rupture with obvious signs and serious consequences, leaving an open communication between the lung and pleural cavity, and invariably resulting in pyopneumothorax, which is usually meant when rupture of lung is spoken of as a complication of artificial pneumothorax.

Of course, spontaneous pneumothorax may occur on the untreated side during collapse therapy and produce bilateral pneumothorax. This happened in two of my cases and both died.

In six of my first 334 cases of artificial pneumothorax (20) rupture of the visceral pleura on the treated side occurred. Two of these were non-tuberculous patients, and both recovered. The other four were cases of pulmonary tuberculosis and all died ; a pyopneumothorax developed in each case. Of Forlanini's 139 cases, rupture of lung occurred in eight, and all died.

Burland (21) describes nine cases. Of these three died within the first month and four in from three to six months.

It must, therefore, be regarded as a very serious complication, which not only justifies, but necessitates, drastic treatment. When the rupture occurs in a collapsed lung the perforation tends to remain open, whereas in an ordinary case of spontaneous pneumothorax the collapsing lung tends to close the perforation and allow healing to take place. The treatment, therefore, is usually that of spontaneous pneumothorax where the perforation does not heal, and is described in Chapter VI. It may here be summarised as follows :—

1. Relieve urgent dyspnœa by removing gas from the pleural cavity and, if necessary, leave in the cavity a needle connected with a rubber tube, the lower end of which is in a bowl of water on the floor, thus allowing gas to leave, but not to enter, the pneumothorax cavity.

2. Clean out the pleural cavity by aspiration and, if necessary, by washing it out with 1 in 5,000 solution of methylene blue.

3. Restore the general health as far as possible by glucose, tonics, fresh air, etc.

4. If the perforation remains open and the lung shows no sign of re-expanding, thoracoplasty should be performed early, as soon as the patient has recovered from the initial shock and when his general condition is good. The longer operation is delayed, the less will be the chance of a successful result.

Thickened Pleura.—This may occur when there is no effusion, but is especially frequent in cases of long standing pyopneumothorax. The collapsed lung may be so bound down by pleura that re-expansion is impossible, and in that case, whether there is an effusion or not, the lung is permanently useless and the pneumothorax cavity a source of possible danger, so it should be closed by thoracoplasty.

After pneumothorax treatment has finished, the visceral and parietal pleura are usually adherent and thickened, so that it is not possible to re-collapse the lung. This is not always the case, however, and I have re-collapsed a lung seven months after the last refill. Vere Pearson (9) succeeded in reproducing a pneumothorax in one case two years after the last refill.

Pleurisy.—A small lesion of pleurisy frequently leads to the pleural layers becoming adherent and the disease arrested. In cases of pneumothorax this cannot occur, since the visceral and parietal pleura are separated by gas, and consequently pleural effusion is a very common complication. Dry pleurisy may occur as a complication of artificial pneumothorax, and Riviere thinks it is not uncommon at the site of puncture, especially if many punctures have been made at the same place. The symptoms of dry pleurisy are malaise, slight pain or aching in the chest, and there is often a little tenderness on palpation over the inflamed pleura. If a refill is given it is found that less gas than usual is required to reach the former pressure. The treatment of such cases is to keep the patient in bed and

apply antiphlogistine to the side. Saugman believed that salicylates have a specific effect in these cases.

Pleural effusion is the most frequent complication of artificial pneumothorax. There may be no symptoms or the patient may complain of malaise and slight pain. Sometimes there is vomiting, and the condition resembles an acute gastric disturbance. As a rule there is some pyrexia for a few days. After three or four days, however, the symptoms usually disappear, but occasionally the temperature remains high and the patient's condition becomes steadily worse. In these latter cases it is found that the tuberculosis is spreading in the lungs, and the development of the liquid seems to have been the starting-point of a general breakdown, though, fortunately, such cases are rare. The physical signs of effusion are obvious: shifting dullness can be demonstrated even if there is only a very small amount of liquid: the level of the liquid is horizontal, and there is no parabolic curve: vocal fremitus is absent over the liquid, but present over the air-space above it: breath sounds are usually absent, but often acquire a metallic quality, especially over a small zone just above the level of the liquid. The coin sound is usually well heard, and the succussion splash is one of the best physical signs, and can be heard even in the smallest effusions. The patient will often complain of feeling liquid splashing about in his chest.

X-ray shows the presence of liquid beyond doubt, but the patient must be examined in the upright position. A very small effusion can often be detected only by X-ray examination.

Various explanations have been given to account for the frequency of pleural effusion in cases of pneumothorax. Irritation by the gas, its temperature, the repeated punctures of the parietal pleura or the intrapleural pressure have all been described as playing a part in the production of effusion. In my opinion, however, these effusions are true tuberculous exudates due to tuberculous pleurisy, and in my

experience they occur with equal frequency whether the gas is heated or not, and whether air, nitrogen, carbon-dioxide or oxygen is used, and in support of this view I would urge the following considerations :—

1. Pleurisy is a common complication of pulmonary tuberculosis and often leads to adherent pleura. If the parietal and visceral pleura are separated, as they are in cases of pneumothorax, one would expect effusion to form when pleurisy occurred. In almost every case of tuberculous spontaneous pneumothorax (where, of course, the disease has spread to the pleura) effusion develops. This is not so in non-tuberculous cases. Osler describes a case where no effusion formed and a quick recovery occurred in a patient who had no signs of tuberculosis and did not react to tuberculin.

2. The cytology of the fluid is that of a tuberculous exudate and not of a transudate.

3. Tubercle bacilli are not found in the early stages in the effusion, but are almost invariably present in long standing cases, and no other organisms have been found in any of my cases.

4. Of the first 309 cases in which I have produced a successful pneumothorax for pulmonary tuberculosis, a definite effusion formed in 128, but of my first fifty-four cases of non-tuberculous disease where pneumothorax was induced, an effusion occurred in one only, and that was only a transient and small effusion in a case of acute pulmonary abscess. A percentage of 41·4 for tuberculous and under 2 for non-tuberculous cases.

Hutchinson (22) thinks that effusion in tuberculous cases is often the result of a small rupture of lung, and supports his opinion by the facts that an effusion is often preceded by a rise in intrapleural pressure, and that the temperature which accompanies its development is very similar to that seen in ordinary cases of spontaneous pneumothorax.

A small transient effusion is almost the rule in cases of

artificial pneumothorax, and if every case were examined daily with the X-ray screen it would be seen in most cases at some time or other as a small pool lying in the costo-diaphragmatic angle. Such small collections of liquid, however, are soon absorbed and are not important. Here I include as cases of pleural effusion only those where it lasts at least a week and covers the diaphragm.

Pleural effusion is not a serious complication of collapse therapy and is often beneficial. It sometimes happens that a patient, who is not doing well, starts to improve as soon as an effusion forms, and eventually makes a good recovery.

The following table shows the results of effusion on a series of 309 cases from my series (23). The cases are those with medium resistance who have failed to improve after other treatment ; C1 included those with unilateral disease, C2 those with slight involvement of the other lung and C3 where there is involvement of a third or more of the better lung :—

	C1.			C2.			C3.		
	Cases.	Died.		Cases.	Died.		Cases.	Died.	
			Per cent.			Per cent.			Per cent.
Total . .	126	24	19·0	135	47	34·8	48	33	68·7
Clear fluid .	36	5	13·9	45	14	31·2	11	8	72·7
No effusion .	78	13	16·6	72	22	30·6	31	22	71·0
Pus . .	12	6	50·0	18	11	61·8	6	3	50·0

From which it will be seen that the development of clear effusion does not increase the mortality, but that when the fluid is purulent the outlook is much more serious. When there is extensive disease the mortality is high in all cases.

Saugman (7) gives the following table :—

	With effusion.		Without effusion.	
	No.	Per cent.	No.	Per cent.
Able to work	31	37·3	24	40·0
Unable to work	4	4·8	1	1·6
Dead	48	57·8	32	53·3
Ill or dead from other causes	—	—	3	5·0

Here, too, there is very little difference between the dry cases and those with effusion.

The effusion usually develops in the first six months of treatment, and after twelve months it is rare.

The next table shows the number of months after the induction of artificial pneumothorax when the effusion formed in my series of 309 cases :—

1 to 3 months	45 cases
3 to 6 ,,	43 ,,
7 to 12 ,,	29 ,,
13 to 18 ,,	5 ,,
Over 18 ,,	6 ,,

When fluid forms, a lung which was previously partially expanded will become completely collapsed. This is very constant, and is one reason why Hutchinson (22) thought effusion in these pneumothorax cases was often due to ruptured lung and consequently increased intrapleural pressure.

In some cases, however, there is no rise of intrapleural pressure when the effusion forms and yet the lung becomes completely collapsed. It may be that the weight of the liquid coming into contact with all parts of the lung as the patient lies down or changes his position presses the air out of the alveoli whatever the intrapleural pressure may be.

When the lung contains some air re-expansion is much easier than in the case of a lung that is completely airless.

Jacot in an extensive study (24) of purulent effusion complicating artificial pneumothorax, recognised four types of cases.

1. In 36 per cent. of his cases the serous liquid developed into tuberculous pus, but the patient's condition remained good.

2. In 16 per cent., after a period of wellbeing, the patient's condition suddenly became grave, there was a hectic temperature and the pulmonary tuberculosis rapidly spread to a fatal issue.

3. In 28 per cent. the purulent effusion began with pyrexia, and the patient's condition was bad, but eventually improved and the case became the same as in the first type.

4. In 20 per cent. the purulent effusion followed rupture of lung and all these cases were fatal.

The treatment of pleural effusion complicating artificial pneumothorax depends on many factors, and each case must be taken on its merits. If the fluid is left it will usually cause obliteration of the pneumothorax cavity by pleural adherence and fibrosis, starting at the bottom of the pleural cavity and working upwards. If this result is desired the fluid should be left, but the patient must be kept under observation lest complications arise, such as pyrexia, change of the fluid to pus, displacement of the mediastinum. Many cases of pneumothorax which terminate by obliteration in this way do very well, for, as the lung is drawn out, a satisfactory fibrosis develops over the diseased areas and the healthy parts of the lung begin to function once more.

If, however, it is decided to maintain the pneumothorax, the effusion must be aspirated and replaced by air to prevent obliteration of the pneumothorax cavity. Usually after one or two aspirations the fluid does not reform.

In replacing fluid the quantity of gas required is less than that of the fluid removed; roughly speaking, about a

quarter the quantity is sufficient. In one case 225 c.cm. of fluid was removed and 200 c.cm. of air given, the pressure being changed from −8 −0 to 0 + 6.

In another case the initial pressure was −17 −9. After removing 600 c.cm. of fluid it became −22 −13, and after another 200 c.cm. had been removed it was −28 −15. On adding 200 c.cm. of air the pressure at once rose to −16 −9, another 200 c.cm. of air made it −10 −6, and after a further 200 c.cm. it became −4 −0. Thus the removal of 800 c.cm. of fluid lowered the pressure from −17 −9 to −28 −15, but the addition of 600 c.cm. of air raised it to −4 −0.

The methods of fluid replacement and washing out the pleural cavity have been described in Chapter VI. I prefer to use two needles in most cases so that air can enter through one as the fluid leaves through the other, and by this means the intrapleural pressure can be kept constant, raised or lowered at will. It is not wise to let the pressure alter too quickly, and too low a pressure is apt to cause symptoms which are sometimes very severe, such as dyspnœa, faintness or paroxysm of cough.

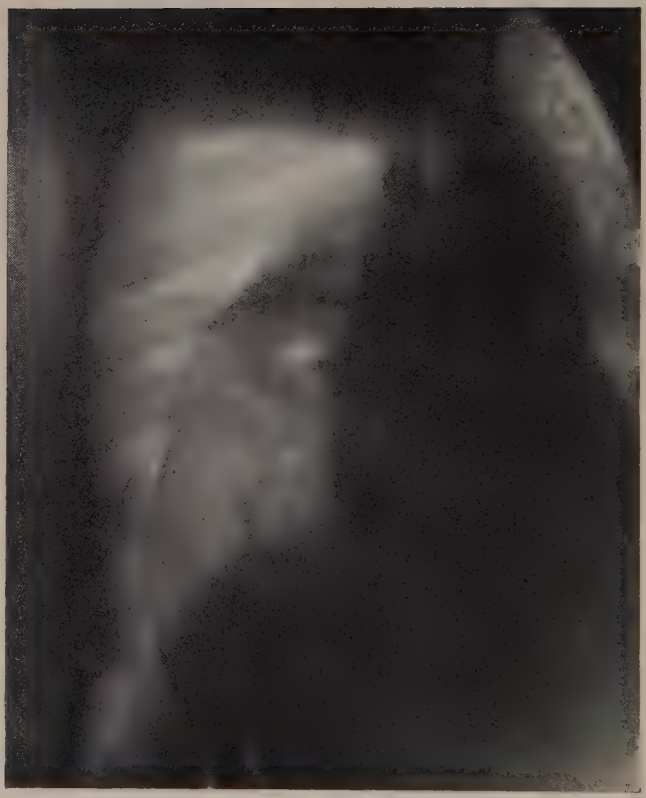
If the fluid is purulent the case is very much more serious, although I have had several patients with large purulent effusions who made good recoveries. The pus should be aspirated and the pressure kept as low as possible in order to help the lung re-expand. If the lung does re-expand the outlook is hopeful, and a satisfactory termination of the treatment by obliteration of the pneumothorax cavity may be expected. If, however, there is no sign of re-expansion the patient will probably have a permanent pyopneumothorax, and be liable to any of the various complications of that condition. In such a case thoracoplasty should be performed as soon as possible, if not contra-indicated by the condition of the other lung. In some cases it may be necessary to wash out the pleural cavity first to get it in as clean a condition as possible ; 1 in 4,000 solution of methylene blue

PLATE XXIX

HYDROPNEUMOTHORAX WITH THE PATIENT LYING ON THE HEALTHY SIDE. NOTE THE DISPLACEMENT OF VISCERA AND POSITION OF THE FLUID LEVEL.

[*To face p.* 196.

PLATE XXX



The Same Case as Plate XXIX with the Patient standing on his Head. The fluid has gravitated to the apex, and the base of the lung and diaphragm are visible.

may be used for this purpose. A preliminary operation for phrenic evulsion may also be performed and some surgeons do this as a routine before the major operation. Since the lung cannot expand, it is permanently useless, and therefore thoracoplasty cannot affect its function. The object of the operation is to close the pleural cavity and not to immobilise the lung as in other conditions.

The Effect of Artificial Pneumothorax on Vital Capacity

Case 1.—Male, twenty-eight. Onset of pulmonary tuberculosis, March, 1919. Sanatorium treatment, July to December, 1919. March, 1920, tubercle bacilli present. Signs of active disease at the apices of the upper and lower lobes on the left side. Vital capacity, 2,570 c.cm. Percentage, — 41.

July.

V.C., 2,450 c.cm. Percentage, — 44.

August 23rd.

V.C., 2,250 c.cm. Percentage, — 48·5.

August 27th.

Artificial pneumothorax. 900 c.cm. of air introduced, the end pressure, — 1. An hour later: V.C., 1,560 c.cm. Percentage, — 65.

August 30th.

Before the first refill. Pressure, — 6·5.
V.C., 2,050 c.cm. Percentage, — 53.
One hour after refill of 900 c.cm. of air and pressure, — 2.
V.C., 1,680 c.cm. Percentage, — 61·6.

September 2nd.

Before the second refill. Pressure, — 5.
V.C., 1,900 c.cm. Percentage, — 56·5.
One hour after refill of 800 c.cm. and pressure 0.
V.C., 1,400 c.cm. Percentage, — 68.

This case illustrates the effect of large initial doses. The patient's vital capacity was getting gradually worse before treatment. 900 c.cm. of air reduced the vital capacity by 690 c.cm. Three days later the vital capacity increased 490 c.cm. and fell 370 c.cm. on the addition of 900 c.cm. of air into the pleural cavity. Three days later it rose 220 c.cm., and was reduced 500 c.cm. by a refill of 800 c.cm.

Case 2.—Male, aged nineteen. Two years' history of pulmonary tuberculosis. Signs of extensive disease of the right lung. Tubercle bacilli present.

September 2nd.

Before artificial pneumothorax.

V.C., 2,380. Percentage, — 45·3.

September 3rd.

Twelve hours after 700 c.cm. of air had been introduced. End pressure, — 2·5.

V.C., 1,800. Percentage, 58·7.

September 6th.

Seventy-two hours after the first refill. Pressure, — 4.

V.C., 2,450. Percentage, — 43·6.

Two hours after 600 c.cm. of air had been introduced.

Pressure, — 0·5.

V.C., 1,700. Percentage, — 60·8.

September 8th.

Forty-eight hours since the second refill.

V.C., 2,050. Percentage, — 53.

September 9th.

Two hours after the third refill of 900 c.cm. Pressure 0.

V.C., 1,500. Percentage, — 65·5.

Case 3.—Male, aged twenty-three. After one year's pneumothorax treatment for pulmonary tuberculosis and four weeks since the last refill. Intra-pressure, — 6.

V.C., 2,180. Percentage, — 52.
One hour after a refill of 1,200 c.cm. Pressure, + 8.
V.C., 1,700. Percentage, — 62·8.
Again four weeks since last refill. Pressure, — 5.
V.C., 2,440. Percentage, — 46·5.
One hour after a refill of 1,500 c.cm. Pressure, + 9.
V.C., 1,570. Percentage, — 65·6.
Again four weeks since last refill. Pressure, — 1.
V.C., 2,100. Percentage, — 54.
One hour after a refill of 800 c.cm. Pressure, + 8.
V.C., 1,680. Percentage, — 63·2.
Again four weeks since last refill. Pressure, — 4·5.
V.C., 2,410. Percentage, — 47.
One hour after a refill of 1,300 c.cm. Pressure, + 5.
V.C., 1,720. Percentage, — 63.
Again four weeks since last refill. Pressure, — 4.
V.C., 2,310. Percentage, — 49·4.
One hour after refill of 1,300 c.cm. Pressure, + 4·5.
V.C., 1650. Percentage, — 64.

From this it would appear that an intrapleural pressure of — 4·5 is the optimum pressure. Pressures of — 6 and — 1 give a worse vital capacity.

Case 4.—The following two cases illustrate the re-expansion of lung. Male, aged fifty-one. After over two years of pneumothorax treatment for pulmonary tuberculosis.

Before a refill.
V.C., 1,870. Percentage, — 46·7.
Three months after completion of treatment.
V.C., 2,480. Percentage, — 29·1.

Case 5.—Male, aged thirty-one. After two years' pneumothorax treatment for pulmonary tuberculosis and six weeks since the last refill. Intrapleura pressure, — 8.

V.C., 3,070. Percentage, — 49·9.
One hour after refill of 1,100 c.cm. Pressure, + 7.

V.C., 2,550. Percentage, — 55·5.
Again six weeks since last refill. Pressure, — 8.
V.C., 3,220. Percentage, — 44·6.
One hour after refill of 1,100 c.cm. Pressure, + 10.
V.C., 2,690. Percentage, — 53·5.
Five months after completion of treatment.
V.C., 3,490. Percentage, — 39·5.
Eight months after completion of treatment.
V.C., 3,780. Percentage, — 34·5.

Case 6.—Male, aged thirty. Infiltration of the upper and lower apices of the left lung. Tubercle bacilli present.

Before pneumothorax treatment was started.
V.C., 2,660. Percentage, — 22·2.
After a year's treatment. Intrapleural pressure, — 17.
V.C., 2,200. Percentage, — 35·6.
An hour after removing 500 c.cm. of liquid and introducing 1,000 c.cm. of air. Pressure, + 13.
V.C., 2,080. Percentage, — 39.
Six weeks later. Pressure, — 16.
V.C., 2,360. Percentage, — 31.
An hour after a refill of 500 c.cm. of air. Pressure, + 18.
V.C., 2,210. Percentage, — 35·5.

Here great changes in pressure cause a very slight alteration in the vital capacity, probably because the lung was fully collapsed by the liquid.

Analysis of Cases treated by Artificial Pneumothorax

Dr. Riviere (13, p. 231) refers to the dangers of judging the results of any form of treatment in cases of pulmonary tuberculosis. He points out that no two cases are exactly alike, and that the methods of classification and analysis

of the cases by different authors vary considerably, so that it is difficult to compare one series with another. However, he published the results of treatment of a large number of cases by various authors, and, although these figures do not really help one to form an opinion as to the value of pneumothorax treatment, they do go to show that a large number of patients who have had the treatment are well enough to work many years later.

Tables giving the results of any treatment in pulmonary tuberculosis are always misleading, because it is impossible to decide how much of the effect—good or bad—was due to the special treatment under review and how much to routine or other methods. Moreover, the chances of a relapse vary enormously according to certain factors, such as the patient's home condition, habits, temperament, means, etc.

One should also remember that certain methods of treatment are only employed if the patient is doing badly. Artificial pneumothorax, for example, is practically never induced in the case of a patient who has early disease and appears to be getting on well without it.

The following figures, therefore, are not intended to illustrate the results of artificial pneumothorax as compared with the results of other methods of treatment, but they do show the types of case for which the treatment was employed and the chances of certain dangers and complications.

The series consists of the first 500 cases in which I induced or attempted artificial pneumothorax for pulmonary tuberculosis. During the same period there were seventy-one cases of non-tuberculous disease and twenty-six cases of spontaneous pneumothorax. They were all treated between the years 1918 and 1926. Including unsuccessful attempts to induce a pneumothorax and refills, the pleura was punctured 10,141 times in the series.

The following accidents occurred from these 10,141 punctures :—

1 case of fatal pleural shock.

3 cases of collapse and alarming symptoms, but recovery without bad after effects.

17 cases of faintness or mild shock.

3 cases of bad surgical emphysema ; all recovered.

57 cases of slight local surgical emphysema.

4 cases where a large vessel or the heart was punctured, and blood rushed up the pneumothorax tubing, but no ill-effects followed.

2 cases had what I thought to be mild gas embolism, but both quickly recovered.

A sinus developed along the track of the needle in five cases with tuberculous pyopneumothorax. In no other type of case was there any infection of the needle track.

During the treatment there were the following complications in this series :—

Spontaneous pneumothorax on the opposite side occurred in three cases and proved rapidly fatal.

Four patients had operation for appendicitis during the treatment and one died.

Six patients were confined during the treatment. In one an acute exacerbation of disease occurred in the untreated lung, but the other five suffered no ill-effects.

Of the 500 tuberculous cases an effusion developed in 177. In 123 of these it was clear and in 54 tuberculous pus. In no case was any organism found except the tubercle bacillus.

Of the non-tuberculous cases in this series an effusion developed in one only, and this was a case of acute abscess where some clear fluid formed, but was very small in amount and was quickly absorbed.

In 46 of the 500 cases a pneumothorax could not be induced owing to adherent pleura, of the remaining 454 the

pneumothorax was induced at the first attempt in 429, at the second in 19, and at the third or fourth in 8.

249 were left-sided, 239 right-sided, and 12 bilateral cases. No difference in result was noticed, whether the case was right- or left-sided.

244 were males and 256 females.

Of the 71 non-tuberculous cases artificial pneumothorax was induced or attempted for the following reasons :—

Bronchiectasis	23
Abscess of lung	14
Recurrent hæmoptysis	8
Pain of pleurisy	5
Replacing pleural effusion	9
Diagnosis	12

Of the 26 cases of spontaneous pneumothorax 17 developed tuberculous pyopneumothorax, and of these 14 died. Of the 3 who are not dead 2 had thoracoplasty. 9 did not develop purulent effusion, and only 2 of these are dead.

I divided the tuberculous cases into the following groups:—

A. Acute pulmonary tuberculosis.

C. Cases with medium resistance who failed to get on under other treatment.

1. Unilateral disease.
2. Slight involvement of the other lung.
3. A third or more of the better lung involved.

The following table shows the number in each group with the number of deaths :—

	Total Cases.	No. Dead.	Male.	Female.
A.	16	9	4	12
C. 1	163	43	83	80
C. 2	173	71	91	82
C. 3	58	50	28	30

In 44 cases there was too much adherent pleura to allow a sufficient collapse to produce any effect, and of these 32 are dead.

In 46 cases no collapse at all could be obtained, and of these 32 are dead.

The following table shows the incidence of effusion in the four groups :—

	Total Cases.	Clear Effusion.	Pus.
A.	16	5 (Of these 3 are dead.)	0
C. 1	163	50 (Of these 13 are dead.)	19 (Of these 13 are dead.)
C. 2	173	56 (Of these 22 are dead.)	25 (Of these 23 are dead.)
C. 3	58	12 (Of these 10 are dead.)	10 (Of these 10 are dead.)

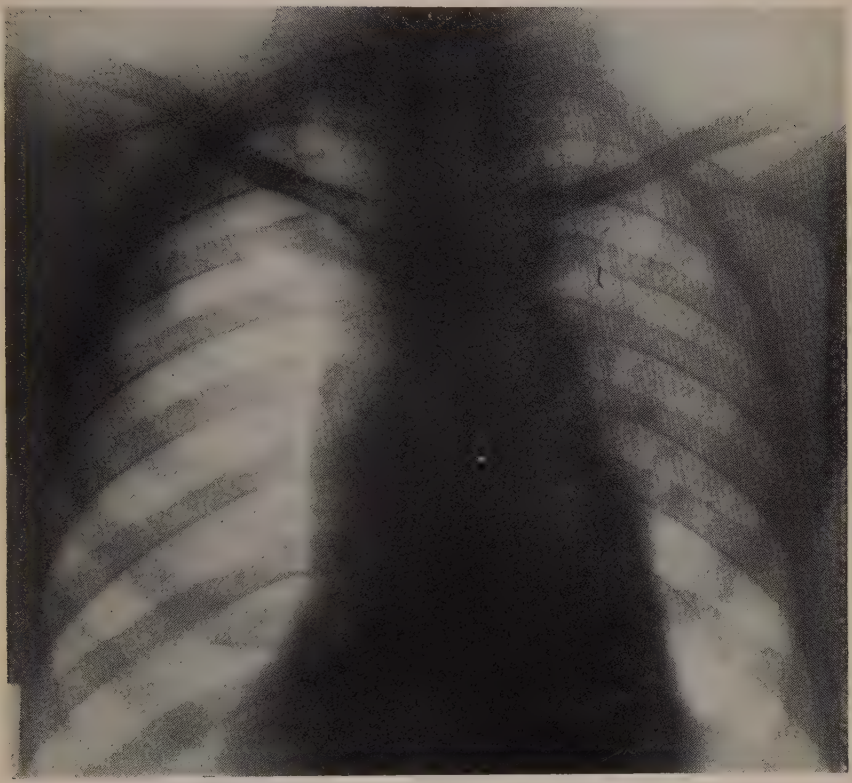
The next table shows the number of months from the onset of treatment to death in those in this series who died :—

Months.	0–6.	6–12.	12–18.	18–24.	24–36.	36–48.	48–60.	Over 60.
A.	6	2	1	—	—	—	—	—
C. 1	1	1	4	2	11	10	9	5
C. 2	3	3	9	15	22	12	4	3
C. 3	10	15	7	12	5	—	—	1

From this analysis the following conclusions may be drawn :—

1. That artificial pneumothorax is a safe method of treatment. Complications may occur, but are rare and certainly no greater than those which may happen during other methods of treatment. Indeed, pneumothorax tends to prevent certain complications—for example, no case of fatal hæmoptysis occurred in my series during the treatment.

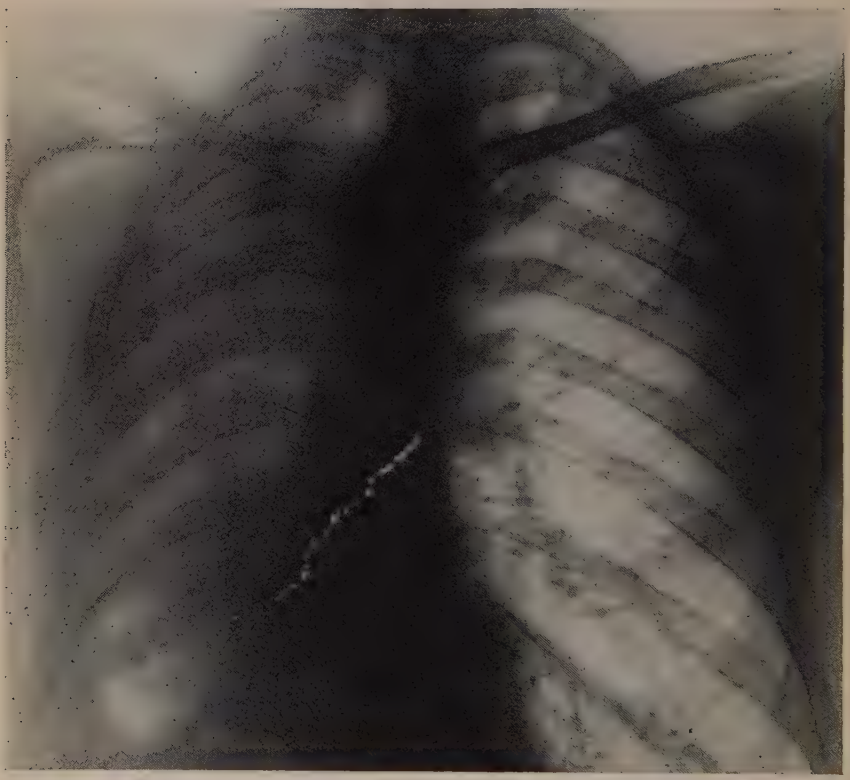
PLATE XXXI



Complete Collapse of Right Lung during Artificial Pneumothorax Treatment, 1923.

[*To face p.* 204.

PLATE XXXII



THE SAME CASE AS PLATE XXXI, BUT TAKEN IN 1928. THE PATIENT IS NOW WELL AND AT WORK. NOTE THE COMPLETE RE-EXPANSION OF THE RIGHT LUNG, WITH FIBROSIS AND DISPLACEMENT OF THE HEART.

2. Unilateral cases do much better than bilateral ones, and when there is much disease in the untreated lung pneumothorax has no effect except that it may prolong life for a short time.

3. The development of a clear effusion does not affect the patient's chance of recovery. The effusion is a tuberculous exudate, and does not occur in the non-tuberculous cases.

4. The development of tuberculous pus, however, makes the outlook very serious.

5. In the majority of cases life is prolonged by artificial pneumothorax.

Drs. R. C. and R. W. Matson and Bisaillon (26) describe the results of 492 cases of pulmonary tuberculosis treated by artificial pneumothorax. They exclude all cases then under treatment or treated within the last two years, so as to show the later rather than the immediate results. They admit that certain factors influence the result; for example, the environmental and social conditions of the patient, his temperament and willingness to give loyal co-operation in the treatment, etc., but under favourable conditions they believe the end result depends on :—

1. The type of the disease.
2. The character of the pneumothorax.
3. The condition of the opposite lung.

They divide their cases into five groups :—

1. Chronic fibro-caseous disease progressive in character, but with little or no cavitation.

2. Chronic fibro-caseous disease more advanced than the first group, progressive in character and with demonstrable cavitation.

3. Chronic fibro-caseous disease with cavities, but not progressive.

4. Acute pulmonary tuberculosis. All were cases of rapidly-advancing disease.

5. Chronic bilateral cases with cavities in both lungs.

	Cases.	Clinically well.	Arrested.	Dead.
		Per cent.	Per cent.	Per cent.
Group I. . . .	194			
Satisfactory collapse	99	53	15	21
Partial collapse .	62	17	19	46
No collapse . .	33	15	12	54
Group II. . . .	177			
Satisfactory collapse	71	40	32	15
Partial collapse .	70	11	17	48
No collapse . .	36	5	8	61
Group III.. . .	52			
Satisfactory collapse	21	43	24	28
Partial collapse .	22	13	9	66
No collapse . .	9	0	22	44
Group IV. . . .	39			
Satisfactory collapse	13	46	7	23
Partial collapse .	17	6	6	76
No collapse . .	9	0	0	97
Group V. . . .	30			
Satisfactory collapse	7	42	0	57
Partial collapse .	12	8	16	58
No collapse . .	11	0	18	63

REFERENCES

(1) Heaf, F. R. G. *Lancet*, 1926, *i.*, 1264.

(2) Rist, E., and Strohl, A. *Études sur le pneumothorax, Ann. de Méd.*, 1920, **VIII.**, 233.

(3) Tobiesen, T. *Beitz. z Klin. d. Tuberk.*, 1911, **XIX.**, 451, and **XXI.**, 109.

(4) Burrell, L. S. T., and MacNalty, A. S. *Med. Res. Council Spec. Report Series*, No. 67.

(5) Forlanini, C. *Gazz. med. ital.*, **XLII.**, 41, 51, 61, 71.

(6) Burnand, R. *Bull. et mém. Soc. méd. d. hop. de Paris*, 1920, **XLIV.**, 1569.

(7) Saugman, C. *Paris Méd.*, 1921, **XLII.** (Annexe), 59 ; *ibid. Lancet*, 1920, *ii.*, 148.

(8) Minor, C. L. *Am. Rev. Tub.*, 1917, **I.**, 522.
(9) Pearson, Vere. *Lancet*, 1919, *ii.*, 148.
(10) Burrell, L. S. T. *Tubercle*, 1925, **VI.**, 161.
(11) Morgan, Parry. *Lancet*, 1913, *ii.*, 18.
(12) Barlow, N., and Kramer, D. *Am. Rev. Tub.*, 1922, **VI.**, 75.
(13) Riviere, Clive. "The Pneumothorax and Surgical Treatment of Pulmonary Tuberculosis," London, 1927, p. 140.
(14) Lawson, J. *B.M.J.*, 1922, *i.*, 309.
(15) Lillingston, C. *Lancet*, 1918, *ii.*, 739.
(16) Jacobæus, H. C. *Am. Rev. Tub.*, December, 1922, 871.
(17) Woodcock, H. de C. *Edinb. M.J.*, 1915, **XV.**, 314.
(18) Stivelman, B. *N. York M.J.*, 1919, **CIX.**, 187.
(19) Hudson, B. *B.M.J.*, 1920, *i.*, 15.
(20) Burrell, L. S. T. *Lancet*, 1924, *ii.*, 167.
(21) Burland, M. *Ann. d. Méd.*, 1921, **IX.**, 22.
(22) Hutchinson, R. C., and Blair, L. G. *Tubercle*, 1926, **VII.**, 417.
(23) Burrell, L. S. T. *B.M.J.*, 1926, *ii.*, 8.
(24) Jacot, M. *Rev. Méd. de la Suisse rom.*, 1915, **XXXV.**, 117, 185, 241.
(25) Bjure, A. *Upsala Läkaref. Förh.*, 1920, **XXV.**, 5.
(26) Matson, R. C., Matson, R. W., and Bisaillon, M. *Tubercle*, 1925, **VII.**, 12.

CHAPTER XII

TREATMENT—*Contd.*

Extrapleural Pneumolysis.—When, owing to adherent pleura, an artificial pneumothorax cannot be induced, part of the lung may be collapsed by stripping the parietal pleura from the chest wall in the desired area and packing the space with solid paraffin or wax.

When the apex of the lung is collapsed in this way it is known as apicolysis, and has been advocated in cases of thoracoplasty where further collapse of the apex is required.

If wax is used it tends in time to find its way out through the skin, and in one case the wax worked its way into the bronchial tubes, and was coughed up in pieces many years later. In this patient left artificial pneumothorax had been tried, and failed owing to adherent pleura, and so extrapleural collapse was induced in Switzerland by inserting 800 c.cm. of wax between the parietal pleura and the chest wall. At that time the patient was cachectic and had tuberculous peritonitis in addition to extensive infiltration and cavitation in the left lung. The immediate result was very striking, he lost his toxic symptoms, tubercle bacilli disappeared from the sputum, the peritonitis improved and he was walking about a few months later. He remained fairly well for about seven years, when symptoms began to increase and a left-sided thoracoplasty was performed and caused improvement for some time. He first began to cough up pieces of wax twelve years after it had been inserted, and this continued until his death some two years later.

In this case the treatment undoubtedly checked the rate of spread of the disease and the patient, although suffer-

ing from advanced disease at the time, lived for fourteen years.

In one of my cases where fat had been used in performing an apicolysis, the place became septic and a large abscess formed. It healed, however, and the patient was actually better, the contracting scar tissue having produced a fair collapse of the apex of the lung.

A similar result may be made by stripping the parietal pleura over the diseased area and plugging with gauze which is gradually removed.

In my experience, however, there are very few cases suitable for apicolysis, better results being obtained by thoracoplasty.

Phrenic Evulsion.—Section or evulsion of the phrenic nerve will cause paralysis of one half of the diaphragm, and consequently those movements of the lungs which are controlled by the diaphragm will cease. The operation is, therefore, useful for giving additional rest to the lung.

Formerly it was the custom to divide the phrenic nerve, and this often failed to produce paralysis of the diaphragm owing to the frequent presence of accessory phrenic nerves. Now, however, the nerve is twisted and some six or more inches are removed. In one case Morriston Davies (1) removed 49 cm. (20 in.) of the phrenic nerve, 29 being the trunk, and 20 cm. the terminal filaments. He found recovery of the diaphragm in only 1 out of 105 cases, and in this one the muscle was paralysed and showed paradoxical movements a month after the operation, but five months later it was functioning normally. In this case 4 in. of nerve had been removed.

The indications for phrenic evulsion are :—

1. To rest the lung in cases of tuberculosis. When the disease is basal great improvement follows the operation, and the disease often becomes arrested. In apical tuberculosis the result is not so striking, and in the majority of cases no effect one way or the other is noted. The degree

to which the apex of a lung is affected by paralysis of the diaphragm is a matter of dispute. There is little doubt that the base is more affected than the apex, but cases have been reported where X-ray showed diminution in the size of apical cavities after phrenic evulsion, and most observers admit that the operation does have some effect on the apex of the lung. Practically, however, one may anticipate good results in the rare cases of basal pulmonary tuberculosis, but not if the disease is apical or generalised.

2. To relieve symptoms. A dry, irritating cough or persistent hiccup may be relieved by phrenic evulsion. In some cases the same result may be obtained by injecting alcohol into the nerve.

3. To test the condition of the better lung before thoracoplasty. If after phrenic evulsion the patient has a focal reaction with signs of activity in the other lung, thoracoplasty should not be performed.

4. As a preliminary to thoracoplasty. This has become a routine with many surgeons, and undoubtedly helps to immobilise the lung.

5. To assist an incomplete artificial pneumothorax. The operation is especially valuable when there are adhesions to the diaphragm.

6. To diminish the size of the pleural cavity. When the pleura is thick and the re-expansion of lung very slow or in cases of tuberculous pyopneumothorax phrenic evulsion may be performed, and the rising diaphragm assists in the obliteration of the pleural cavity.

7. To relieve the heart in cases of great displacement in fibrotic cases. Morriston Davies describes six cases where symptoms resulting from fibrosis and adhesions were relieved by the operation.

In a report on 105 cases treated by phrenic evulsion, Morriston Davies (1) comes to the following conclusions :—

1. In true basal cases of tuberculosis it has been unquestionably of immense assistance in arresting the disease ;

but that with one brilliant exception it is of little use in cases of basal bronchiectasis.

2. When the disease is generalised, but mainly unilateral, some improvement, and at times of considerable degree, may be expected in about 50 per cent. of the cases. This improvement has been noticed in cases of bronchiectasis, as well as in those suffering from tuberculosis.

3. As an accessory to incomplete artificial pneumothorax, considerable benefit can be expected in a large percentage of cases, particularly when the base of the lung is adherent to the diaphragm, or it is desired to increase the interval between the refills. Phrenic evulsion should always be done before allowing a lung long collapsed to re-expand.

4. Hemidiaphragmatic paralysis will frequently produce complete relief of distressing symptoms which are due to irritation of the muscle or to distortion, or to drag on the pericardium by adhesions.

5. Phrenic evulsion should always be done as a preliminary to thoracoplasty. On occasion the result of the minor operation is so satisfactory that the major one becomes unnecessary.

6. As bronchiectasis is liable to develop as a sequela of an unresolved pneumonia, phrenic evulsion should be done as a prophylactic measure in all those cases in which resolution is still incomplete three to six months after the onset of the basal pneumonia.

REFERENCE

(1) Davies, H. Morriston. *Tubercle*, 1928, **IX.**, 205.

Thoracoplasty.—Tuberculosis spreads by the lymphatics, and the lymph flow is largely dependent on the movements of the lungs.

The object of thoracoplasty is to immobilise the lung and produce lymph stasis. The procedure differs from artificial pneumothorax in that the collapse of lung is permanent, and it should not be performed, therefore, until the patient has a certain degree of resistance as indicated by fibrosis. In the acute caseating cases, thoracoplasty is contra-indicated, but artificial pneumothorax may be induced, and will often check the disease altogether, or convert it into the chronic fibroid type, in which case thoracoplasty may later be possible.

Some disease in the better lung is present in most cases, and does not necessarily contra-indicate thoracoplasty, although the condition of the better lung is, of course, a matter of far greater importance than if artificial pneumothorax is contemplated. The degree of activity is of more importance than the actual extent of the disease.

The indications for the operation are :—

1. To close the pleural cavity when the lung is collapsed and cannot re-expand. In cases where the general condition of the patient remains good and the pneumothorax is dry, the chest wall will usually fall in naturally ; this, together with compensatory changes in the better lung, will usually close the cavity in time without thoracoplasty, and there is no urgency for the operation. In cases of tuberculous empyema, however, and especially if associated with spontaneous pneumothorax and an open perforation in the visceral pleura, thoracoplasty will give the patient by far the best chance, and it should be performed as soon as it is clear that the lung cannot re-expand, and when the general condition of the patient is at its best.

2. In any case where artificial pneumothorax is indicated but cannot be performed owing to adherent pleura, provided always that

1. Disease (if any) in the better lung is not active.
2. The patient has a reasonable degree of resistance as indicated by fibrosis.
3. Other general contra-indications are absent.

Contra-indications.—Apart from the essentials, namely, power of resistance and absence of activity in the other lung, certain other factors must be taken into consideration. The condition of the heart is important. If there is dyspnœa or other cardiac symptoms due to fibrosis, and consequent displacement of the heart, thoracoplasty may be beneficial, but if the heart muscle is weakened by prolonged tuberculous toxæmia, the operation will be much more serious, and the chances of death from cardiac failure considerable.

The age of the patient must also be considered, and some surgeons think the operation should not be performed in those over forty-five years, although successful cases in patients over fifty have been described. It is, however, more a matter of the general condition and resistance of the patient than his actual age.

Cases of left-sided disease do better than those where the operation is performed on the right side, in some published results the subsequent mortality being twice as great in the right- as in the left-sided cases. In a case, therefore, when the advisability of thoracoplasty is in doubt, the decision for or against the operation may depend on whether the disease is on the right or left side.

Thoracoplasty should not be performed as a last resort. When a patient is desperately ill, emaciated, febrile and toxic, an operation will add considerably to his sufferings. If improvement occurs by other means, such as absolute rest and general treatment, thoracoplasty may be possible later, but if these methods fail the operation could not have succeeded, and most probably would have hastened the end.

Archibald (1) stresses the importance of resistance, and does not operate unless there is evidence of satisfactory fibrosis. Fibrosis causes displacement of the thoracic viscera,

and the presence of the trachea in the mid-line may be taken as a contra-indication for the operation. He advocates thoracoplasty, especially when the most affected lung is tending to heal by fibrosis, but complete healing is prevented by cavitation or insufficient yielding of surrounding tissues. It is an operation for the productive, and not for the exudative type of case.

Of contra-indications, Archibald mentions the following :

1. Several large cavities in both lobes. In this case a very partial and temporary improvement is the best that can be expected, and will not compensate the patient for the suffering of the operation.

2. Activity in a chronic case, especially if due to spread in the other lung. For these cases artificial pneumothorax, phrenicotomy or absolute rest gives a better chance, and if they do not improve they would not have done so with thoracoplasty. " The operation," he says, " may tumble them on the wrong side of the fence."

3. Acute exudative progressive pulmonary tuberculosis, even if apparently unilateral. Here a pneumothorax should be induced.

He advocates resection of the ribs from the first to eleventh near the angles. Collapse is produced by the inward spring of the rib stumps and the natural elasticity of the lung. Those movements of lung which depend on the ribs are stopped, and the lung can be still further immobilised by cutting the phrenic nerve and stopping the lung movements dependent on the diaphragm.

The technique of the operation varies with different surgeons, and for descriptions of it the reader is referred to the works of Lilienthal (2), Alexander (3) and Morriston Davies (4). A large number of patients have now been treated, and the mortality of the operation has been so much reduced and the results so greatly improved, that in skilled hands thoracoplasty need no longer be regarded as a dangerous and almost hopeless procedure.

Discussing the after effects of surgical procedures in cases of pulmonary tuberculosis Tudor Edwards (5) gives some interesting figures showing the results of thoracoplasty.

REFERENCES

Thoracoplasty.

(1) ARCHIBALD, E. *Canadian Med. Ass. Journal*, 1928, p. 3.
(2) LILIENTHAL, H. "Thoracic Surgery," Philadelphia and London, 1925.
(3) ALEXANDER, J. "The Surgery of Pulmonary Tuberculosis," London, 1925.
(4) DAVIES, H. MORRISTON. "Surgery of the Lung Pleura," London, 1919.
(5) EDWARDS, A. TUDOR, *B.M.J.*, 1928, **II.**, 602.

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